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RESPIRATORY CARE

A MONTHLY SCIENCE JOURNAL
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SPECIAL ISSUE

TRACHEAL GAS INSUFFLATION: CURRENT STATUS AND FUTURE PROSPECTS

- TGI and Related Techniques to Introduce Gas Flow into the Trachea
- Ventilator-Induced Lung Injury & the Evolution of Lung-Protective Strategies in ARDS
- Animal & Lung Model Studies of TGI
- Clinical Studies of TGI
- Complications of TGI
- Catheters for TGI
- Monitoring and Humidification during TGI
- Intratracheal Catheters As Drug Delivery Systems

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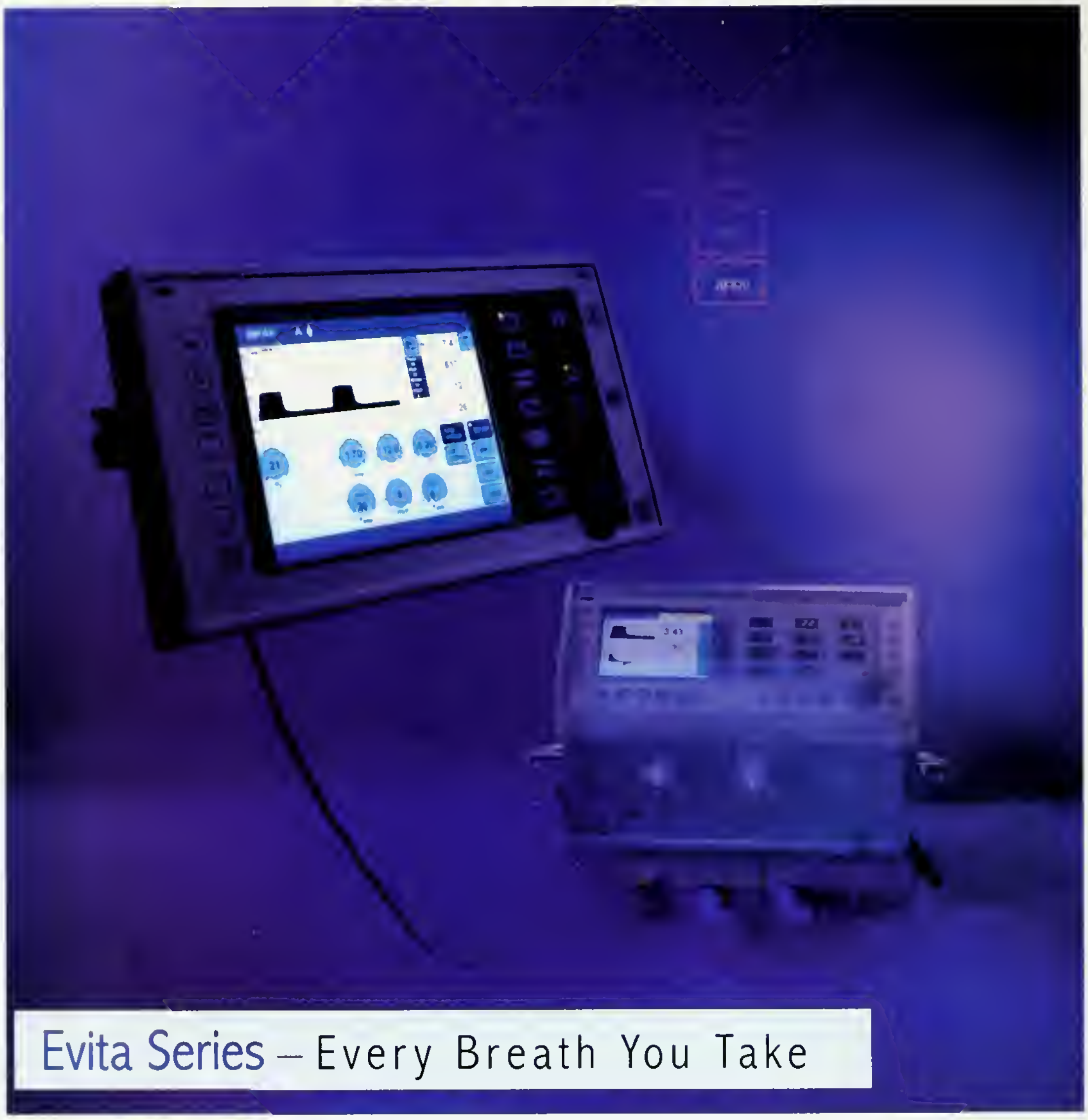

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A SPECIAL ISSUE

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Smoking and Lung Cancer (editorial)-Doll SR. Am J Respir Crit Care Med 2000 Jul;162(1): 4–6.

Progress in Soft Tissue Paleopathology-Aufderheide AC. JAMA 2000 Nov 22/29;284(20): 2571–2573.

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CAPTURE! SHOCK! EXCITE! Clinical Trial Acronyms and the "Branding" of Clinical Research-Berkwits M. Ann Intern Med 2000 Nov 7;133(9):755–762.

For and Against: Why Journals Should Not Publish Articles Funded by the Tobacco Industry. BMJ 2000 Oct 28;321(7268):1074.

Noninvasive Positive Pressure Ventilation in Acute Respiratory Failure-Keenan SP. JAMA 2000 Nov 8;284(18):2376–2378. to notes for Feb 2001

Introduction of Sedative, Analgesic, and Neuromuscular Blocking Agent Guidelines in a Medical Intensive Care Unit: Physician and Nurse Adherence—Bair N, Bobek MB, Hoffman-Hogg L, Mion LC, Slomka J, Arroliga AC. Crit Care Med 2000 Mar;28(3):707-713.

OBJECTIVE: To determine physician and nurse adherence with sedative, analgesic, and neuromuscular blocking agent guidelines in the management of mechanically ventilated patients in a medical intensive care unit. DESIGN: Prospective cohort study. SUBJECTS: One hundred consecutively admitted patients to a medical intensive care unit who required mechanical ventilatory support. A sample of 29 nurses, residents, and attending physicians were interviewed regarding their attitudes and perceptions of the guidelines. MEASUREMENT: Data were collected from concurrent medical records and included the following: demographic characteristics; clinical variables; physician prescriptions of sedative, analgesic, and/or neuromuscular blocking agents; nurse administration of these medications; documentation of monitoring; and assessment of patient hemodynamic status and behaviors. A semistructured interview was elicited from both nurses and physicians about their rationale for the use or nonuse of the guidelines. RESULTS: Patients ranged in age from 24 to 87 yrs, mean 60.7 (± 15.3) yrs. Admission Acute Physiology and Chronic Health Evaluation III scores ranged from 36 to 192, mean 93.8 (± 30.5) and median 88. Length of mechanical ventilatory support ranged from 1 to 112 days, mean 14.8 (± 20.0) days, and median 8 days; medical intensive care unit length of stay ranged from 1 to 46 days, with a mean of 9.8 (± 8.1) days and a median of 8 days. Of the 100 patients, 47% died, 28% returned home, and 25% were discharged to a nursing facility. Eighty-five patients were administered one or more sedative, analgesic, and/or neuromuscular blocking agent, range 1-9 drugs, mean 2.5 (± 1.5) drugs. Physicians prescribed 14 different medications; the most commonly administered drug was lorazepam (n = 71), followed by morphine (n = 39). Physicians and nurses had partial or total adherence to the guidelines in 58% of patients. The initial choice of the drug followed the guidelines in 60% of patients; the overall guideline was followed in 23% of patients. The most common rationales for nonadherence to the guidelines stated by both physicians and nurses were patient-specific factors, resident guideline learning curve, and physician medication preferences. CONCLUSION: Most patients required treatment for agitated behaviors. The majority of treatment regimens partially or totally adhered to the guidelines. Factors such as patient-specific disease states, resident guideline learning curve, and physician preferences of medications may have decreased adherence. Improving adherence to the guidelines is essential to assess their effectiveness in improving clinical outcomes.

Predisposing Factors for Nosocomial Pneumonia in Patients Receiving Mechanical Ventilation and Requiring Tracheotomy—Georges H, Leroy O, Guery B, Alfandari S, Beaucaire G. Chest 2000;118(3):767-774.

Study objectives: To assess the incidence of nosocomial pneumonia (NP) after tracheotomy in an ICU population and to determine NP risk factors during the ICU stay, particularly on the day of tracheotomy. Design: A retrospective study using prospectively collected data. Setting: A 16-bed multidisciplinary ICU. Patients: One hundred thirty-five patients requiring tracheotomy for mechanical ventilation (MV) weaning. Results: The mean (± SD) duration of MV before tracheotomy was 17.8 ± 13.4 days. Thirty-seven cases of NP occurred in 35 patients (25.9%), 8.7 ± 7.3 days after the tracheotomy procedure. NP cases were classified as early NP (n = 19) if they occurred within 5 days after the procedure (mean, 2.7 ± 1.1 days), and as late NP (n = 18) if they occurred beyond the fifth day (mean, 14.4 ± 6.1 days). Multivariate analysis identified the following three independent factors associated with early NP: the presence of pos-

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itive endotracheal aspirates (EAs) with pathogen levels of ≥ 105 cfu/mL ($p = 0.0001$); hyperthermia (temperature, ≥ 38.3°C; $p = 0.002$) on the day of tracheotomy; and the continuation of sedation beyond 24 h after the tracheotomy ($p = 0.0001$). Accountable pathogens of early NP were present in EA on the day of tracheotomy ($p = 0.001$). Cases of late NP were significantly associated with the duration of sedation before the procedure ($p = 0.002$) and with hyperthermia (temperature, ≥ 38.3°C) on the day of tracheotomy ($p = 0.0005$). The ICU admitting diagnosis, previous NP, duration of administration of antimicrobial agents and MV before tracheotomy, indication for tracheotomy, P_{O_2}/fraction of inspired oxygen ratio, and use of steroids on the day of the procedure were not associated with the occurrence of NP. The mortality rate of our population was 33.3%, and NP increased this percentage to 54.3%. Conclusions: Our results could suggest that tracheotomy should be delayed in mechanically ventilated patients with bronchial colonization and hyperthermia, when sedation cannot be discontinued after the procedure, to prevent occurrence of early NP.

Formation and Current Results of a Patient-Organized Registry for Alpha$_1$-Antitrypsin Deficiency—Stoller JK, Brantly M, Fleming LE, Bean JA, Walsh J, for the Alpha One Foundation Research Network Registry Investigators. Chest 2000;118(3):843-848.

Background: Significant challenges exist to investigating uncommon illnesses because too few patients are seen at any single clinical center to permit appropriate research studies. Recognizing this impediment to clinical research in alpha$_1$-antitrypsin deficiency, the Alpha One Foundation, a patient-organized research foundation, has collaborated with clinician-scientists to organize a voluntary registry of individuals with alpha$_1$-antitrypsin deficiency. Purpose: To facilitate clinical research in alpha$_1$-antitrypsin deficiency by organizing a registry of affected individuals willing to be approached to participate in clinical studies. Methods: Elements of the Alpha One Foundation Research Network Registry include a Medical and Scientific Advisory Committee, composed of physician-investigators and patient advocates, designated clinical resource centers at medical institutions with expertise in the management of individuals with alpha$_1$-antitrypsin deficiency, and a data coordinating center with responsibility for database management and analysis. Questionnaires requesting information about demographic features, alpha$_1$-antitrypsin phenotype, smoking history, and health-care utilization were distributed to prospective registrants through the following channels: mailings from the Alpha One Foundation; mailings from the clinical resource centers; and distribution by home-care and pharmaceutical companies. Information from this questionnaire formed the basis of the initial registry database. Results: Between May 1997 and June 1999, 7,789 forms were distributed, and forms were returned by 712 unique registrants. Registrants have the following characteristics: mean (± SD) age, 49.3 ± 13.2 years; women, 47.7%; white, 96.2%; PI*ZZ phenotype, 70.7%; ex-smokers, 73.3%; COPD patients, 87.2% (emphysema patients, 54.2%; chronic bronchitis patients, 33%); and self-reported liver disease, 6.4%. The mean number of physician visits reported by registrants in the preceding 12 months was 7.8 ± 9.4, 59% reported currently receiving I.V. augmentation therapy, and 35% reported using supplemental oxygen at home. Examples of ongoing research studies using this unique database include: (1) a case-control study to evaluate occupational risk factors for obstructive lung disease in individuals with alpha$_1$-antitrypsin deficiency and (2) a study to evaluate the health-care costs for affected individuals. Conclusions: A registry currently including 712 individuals with alpha$_1$-antitrypsin deficiency has been organized through a collaboration between physician-investigators and a patient-organized research foundation. Use of the registry has already facilitated studies that were previously difficult because of the paucity of identifiable study subjects. The registry cohort promises to provide an important resource for future clinical and epidemiologic studies.

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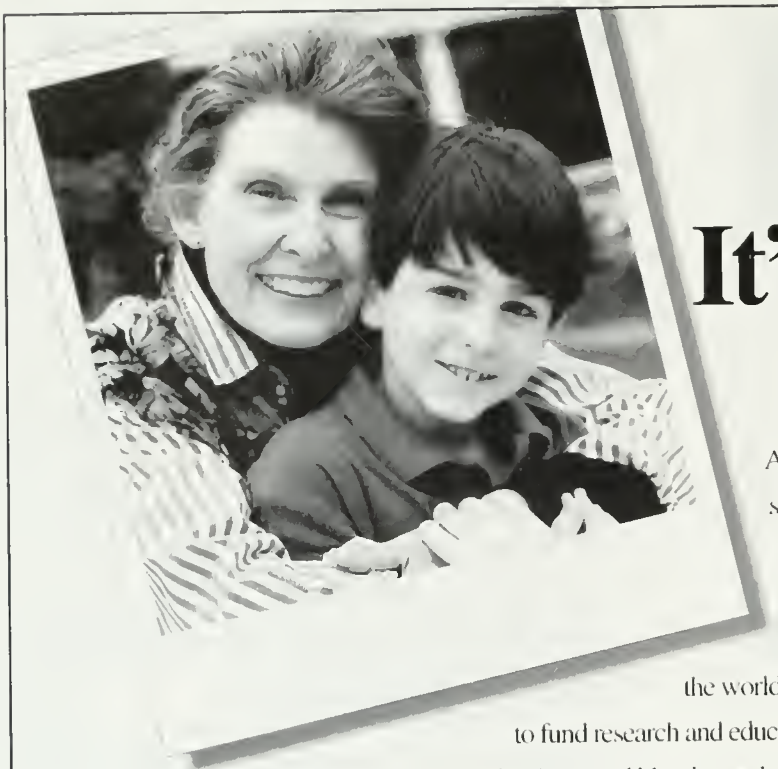

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Evidence for the Need of Bedside Accuracy of Pulse Oximetry in an Intensive Care Unit—Seguin P, Le Rouzo A, Tanguy M, Guillou YM, Feuillu A, Malledant Y. Crit Care Med 2000 Mar;28(3):703-706.

OBJECTIVE: To compare pulse oximetry saturation (S_{po_2}) with arterial blood gas saturation (S_{ao_2}) obtained during clinical routine to determine the optimal lowest reliable value of S_{po_2} in ventilator-dependent patients before setting up a nurse-directed protocol of F_{IO_2} titration. DESIGN: Prospective clinical study. SETTING: Surgical intensive care unit in a university hospital. PATIENTS: Thirty-three patients with a pulse oximeter probe in whom arterial blood gas was measured with a radial artery line. INTERVENTIONS: S_{po_2} was recorded by the nurses and compared with S_{ao_2} obtained by blood gas analysis with a co-oximeter. Two sensors currently used in our surgical intensive care unit and connected to a monitor (HP OmniCare M1165/66A; Hewett Packard, Andover, MA) were tested. In group I, the Durensor DS 100A (Nellcor Puritan Bennett, Pleasanton, CA), a reusable sensor, was used. In group II, the Oxisensor D25L (Nellcor Puritan Bennett), a nonreusable sensor, was used. MEASUREMENTS AND MAIN RESULTS: In group I, 64 data pairs were obtained. In this group, S_{ao_2} ranged from 87 to 98% and S_{po_2} ranged from 92 to 100%. The bias was -1.90% and the limits of agreement ranged from -5.56 to 1.76%. In group II, 47 data pairs were obtained. In this group, S_{ao_2} ranged from 87 to 99% and S_{po_2} ranged from 92 to 100%. The bias was -2.49% and the limits of agreement ranged from -6.62 to 1.64%. CONCLUSIONS: In the range of S_{ao_2} tested, regardless of the sensor used, S_{po_2} overestimated S_{ao_2}. Large limits of agreement were found. Based on this result, the authors concluded that before defining a nurse-directed protocol of F_{IO_2} titration with S_{po_2}, the material used daily must be evaluated. A minimum threshold S_{po_2} value of 96% in both groups I and II is more reliable to ensure $S_{ao_2} \geq 90\%$.

Airway Accidents in Intubated Intensive Care Unit Patients: An Epidemiological Study—Kapadia FN, Bajan KB, Raje KV. Crit Care Med 2000 Mar;28(3):659-664.

OBJECTIVE: To assess the rate of occurrence and nature of airway accidents in intubated patients. DESIGN: Prospective recording of all airway accidents in a 16-bed multidisciplinary intensive care unit. PATIENTS: A total of 5,046 ventilated patients intubated for 9,289 days during 4 yrs. MEASUREMENTS AND MAIN RESULTS: We determined the number and diagnoses of intubated and ventilated patients, the number and timing of airway accidents, the type of tracheal tube used and duration for which the tube was in situ, the description of the type of accident, the severity of the accident, and its impact on the course of the patient's illness, whether the patient needed reintubation, and whether the accident was preventable. The total accident rate was 36 of 5,046 patients during 9,289 intubated patient days; 26 occurred in 5,043 endotracheally intubated patients during 8,446 patient endotracheal tube days. There were 10 tracheostomy-related accidents from a total of 79 patients with tracheostomies during 843 tracheostomy patient days. Six had severe consequences and one resulted in death. Eleven were completely preventable, 17 partly preventable, and 8 were considered unpreventable. Self-extubation was the most common accident. Seven of 13 self extubations occurred in patients due for elective extubation in the next few hours. Twelve of 15 patients with self- or accidental extubation of an endotracheal tube accidents did not require reintubation. CONCLUSIONS: Airway accidents occurred at low levels with even lower rates of resultant morbidity and mortality. Tracheostomy accidents are more common than those with an endotracheal tube.

Safety, Efficacy, and Cost-Effectiveness of Mechanical Ventilation with Humidifying Filters Changed Every 48 Hours: A Prospective,

Randomized Study—Markowicz P, Ricard JD, Dreyfuss D, Mier L, Brun P, Coste F, et al. Crit Care Med 2000 Mar;28(3):665-671

OBJECTIVE: To determine whether three hydrophobic and hygroscopic heat and moisture exchangers (HMEs) retain their heating and humidifying properties (assessed by psychrometric measurements of absolute humidity, relative humidity, and tracheal temperature) for 48 hrs without any drop in their bacteriologic efficiency. DESIGN: Prospective randomized clinical trial. PATIENTS: Sixty-one consecutive unselected mechanically ventilated intensive care unit patients. INTERVENTIONS: Patients were randomly allocated to one of the three HMEs studied (Hygrobac-Dar from Mallinckrodt, n = 21; Humid-Vent from Gibeck, n = 20; and Clear-Thermal from Intersurgical, n = 20). MEASUREMENTS AND MAIN RESULTS: Hygrometric parameters were measured by psychrometry after 3, 24, and 48 hrs of use. Peak airway pressure was recorded every 6 hrs and averaged over 24 hrs. Bacterial colonization of both patients and circuits was studied. Patients in all three groups were similar in terms of age, indications for, and overall duration of mechanical ventilation. Tracheal tube occlusion never occurred. Hygrometric data included 371 measurements whereas bacteriologic data included > 700 samples and cultures. The Hygrobac-Dar HMEs gave a significantly higher absolute humidity whatever the time of measurement (3, 24, or 48 hrs) than the other two HMEs ($p < 0.001$). The Clear-Thermal HMEs gave the poorest hygrometric parameters ($p < 0.01$); five of them were replaced prematurely (24 hrs) because the absolute humidity was < 25 mg H_2O/L. This did not occur for the other HMEs. Mean peak airway pressures were identical in the three groups. The bacterial colonizations of both patient and circuit were similar (and negligible for circuits) for all three groups. CONCLUSION: Some HMEs may be used safely for 48 hrs without change. However, this does not pertain to every brand of HME. Objective in vivo evaluation of their humidifying performances is decisive before extending their duration of use.

A Cost-Effectiveness Analysis of the Application of Nitric Oxide Versus Oxygen Gas for Near-Term Newborns with Respiratory Failure: Results from a Canadian Randomized Clinical Trial—Jacobs P, Finer NN, Robertson CM, Etches P, Hall EM, Saunders LD. Crit Care Med 2000 Mar;28(3):872-878.

OBJECTIVE: To conduct a cost-effectiveness analysis of the use of inhaled nitric oxide (NO) vs. oxygen administered to near-term (gestational age ≥ 34 wks) newborns with severe respiratory illness that were referred for consideration of extracorporeal membrane oxygenation (ECMO). DESIGN: The cost-effectiveness analysis is based on outcome and utilization data from two multicentered randomized clinical trials conducted by the Canadian Inhaled Nitric Oxide Study group, one for patients with congenital diaphragmatic hernia (CDH) and one for patients without CDH. Data from the western Canadian ECMO center were used to establish costs. SETTING: Patients were cared for in Canadian regional neonatal intensive care units, including two ECMO centers. Air transport was used for transporting patients between centers. PATIENTS: Term and near-term newborns with severe respiratory illness who were receiving maximum conventional therapy and whose oxygenation index was > 40. INTERVENTIONS: Patients randomly received NO or oxygen. If their conditions deteriorated, they qualified for ECMO. Not all that qualified for ECMO received it because of individual parent/physician preferences. MEASUREMENTS AND MAIN RESULTS: The cost-effectiveness ratio was the ratio of net cost (including neonatal intensive care, ECMO, and transport) to net outcome (survival) for the two interventions. For non-CDH cases, the cost-effectiveness ratio was $36,613 (Canadian) per life saved; the confidence intervals were wide and the

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results were not statistically significant. For CDH patients, the death rate was lower for oxygen and the oxygen patients cost less; the results were not statistically significant. CONCLUSIONS: The small numbers of patients in the trials precluded significant results. Further, our results have a short-term time horizon (discharge to home or death). Thus, for non-CDH patients, the favorable ratio provides very qualified evidence in favor of NO.

Outcome of Children Who Require Mechanical Ventilatory Support after Bone Marrow Transplantation—Keenan HT, Bratton SL, Martin LD, Crawford SW, Weiss NS. Crit Care Med 2000 Mar;28(3):830-835.

OBJECTIVE: To identify clinically measurable factors that could predict outcome for pediatric patients undergoing mechanical ventilatory support after bone marrow transplant. DESIGN: Cohort study. SETTING: A referral center for bone marrow transplant patients in Seattle, Washington. PATIENTS: Children $<$ 17 yrs old who received a bone marrow transplant and subsequently required mechanical ventilatory support for $\geq$ 24 hrs between 1983 and 1996. INTERVENTIONS: None. MEASUREMENTS AND MAIN RESULTS: Data were abstracted from the charts of 121 pediatric patients who received a bone marrow transplant and subsequently required mechanical ventilatory support. A total of 19 patients (16%) survived to be extubated and survived for $\geq$ 30 days post-extubation. Major risk factors for death included respiratory failure as the reason for endotracheal intubation (4% survival), the presence of pulmonary infection (6% survival), and impairment of more than one organ system (2% survival if more than one organ system was dysfunctional on day 7 postintubation). CONCLUSIONS: Although the prognosis generally is poor among pediatric bone marrow transplant recipients who subsequently require mechanical ventilatory support, there appear to be some groups within this population in whom the likelihood of survival is close to 0. Because the chance of survival was so small for children with dysfunction of more than one organ system on day 7 after intubation, a recommendation to limit medical support for these children could be considered pending the results of other studies.

The Safety of One, or Repeated, Vital Capacity Maneuvers during General Anesthesia—Magnusson L, Tenling A, Lemoine R, Hogman M, Tyden H, Hedenstierna G. Anesth Analg 2000 Sep;91(3):702-707.

A vital capacity maneuver (VCM) (inflating the lungs to 40 cm H_2O for 15 s) is effective in relieving atelectasis during general anesthesia or after cardiopulmonary bypass (CPB). The study was undertaken to investigate the safety of one or repeated VCM. Five groups of six pigs were studied. Two groups had general anesthesia for 6 h and one group received a VCM every hour. Three other groups received CPB. VCM was performed after CPB in two of these groups. VCM was then repeated every hour in one of the groups. Lung damage was evaluated by extravascular lung water (EVLW) measurement, light microscopy, and the half-time ($T_{1/2}$) of disappearance from the lung of a nebulized aerosol containing ^{99m}Tc-DTPA. No changes were noted in extravascular lung water. The pigs subjected to VCM decreased their $T_{1/2}$. In the groups exposed to repeated VCM, $T_{1/2}$ remained lowered (CPB pigs) or decreased over time (non-CPB pigs). No lung damage could be seen on the morphology study. These results suggest that one VCM is a safe procedure. The increase in lung clearance of ^{99m}Tc-DTPA not associated with an increase in lung water when VCM is repeated may have been caused by an increase in lung volume. Therefore, repeated VCM also appears to be safe. IMPLICATIONS: This study demonstrates in an animal model that inflating the lung once or repeatedly to the vital capacity is a safe procedure. This maneuver, also called the vital capacity maneuver, can be used to relieve lung collapse which occurs in all patients during general anesthesia.

Improving Patient Compliance with Asthma Therapy—Chapman KR, Walker L, Cluley S, Fabbri L. Respir Med 2000 Jan;94(1):2-9.

Patients fail to comply with asthma medication for a variety of reasons. These range from physical inability to use an inhaler, through simple forgetfulness, to a conscious decision not to use medication as prescribed due to internal or cultural health beliefs or socioeconomic factors. In some patients, poor self-care because of deep-rooted psychological factors (i.e. factors of which patients have only limited awareness) can affect compliance. Poor doctor-patient communication can be the cause in many other individuals. Thus, there is no single solution that will improve compliance in all patients. Simplifying the regimen or providing memory aids will be sufficient for some patients, while education or psychological counselling will be more appropriate for others. Doctors can also use a range of communication skills to improve the way in which they present information, motivate patients and reinforce progress. These approaches, plus respect for patients' health beliefs and involving them in treatment decisions, can help foster an atmosphere of mutual responsibility and concordance over medicine taking.

Importance of Adjusting Carbon Monoxide Diffusing Capacity (DLCO) and Carbon Monoxide Transfer Coefficient (KCO) for Alveolar Volume—Johnson DC. Respir Med 2000 Jan;94(1):28-37.

The volume dependence of single breath carbon monoxide diffusing capacity (DLCO) and carbon monoxide transfer coefficient (KCO) was determined in 24 healthy subjects. The change in DLCO [fraction of DLCO measured at total lung capacity (TLC)] to change in alveolar volume [fraction of alveolar volume ($\dot{V}_A$) at TLC] closely fitted a simple linear regression and matched a theoretical model. As $\dot{V}_A$ decreased, DLCO fell linearly and KCO increased as expected from the relation of DLCO to $\dot{V}_A$. The equations for adjustment of predicted DLCO and KCO for alveolar volume are: DLCO/DL COtlc = 0.58 + 0.42 $\dot{V}_A/\dot{V}_A$ tlc, KCO/KCOtlc = 0.42 + 0.58/($\dot{V}_A$ /$\dot{V}_A$ tlc). DLCO and KCO were evaluated in 2313 patients. Subgroups of patients with asthma, emphysema, extrapulmonary lung disease, interstitial lung disease and lung resection were identified. Unadjusted DLCO and KCO percent predicted values showed large differences and much variability, so can be misleading. As expected, KCO and DLCO percent predicted values adjusted for alveolar volume were nearly identical. Subgroups have characteristic patterns of $\dot{V}_A$ and unadjusted and adjusted DLCO and KCO. Changes in DLCO and KCO with alveolar volume are relevant for accurate interpretation of diffusion in patients with low lung volumes. Adjusting predicted DLCO and KCO for alveolar volume provides a better assessment of lung function.

Long-Term Effects Nasal Continuous Positive Airway Pressure on Daytime Sleepiness, Mood and Traffic Accidents in Patients with Obstructive Sleep Apnoea—Yamamoto H, Akashiba T, Kosaka N, Ito D, Horie T. Respir Med 2000 Jan;94(1):87-90.

To describe the long-term effects of nasal continuous positive airway pressure (CPAP) on the rate of traffic car accidents, excessive daytime sleepiness (EDS) and mood in patients with obstructive sleep apnoea syndrome (OSAS), we investigated the changes of these parameters before and after nasal CPAP treatment using a questionnaire. Seventy-five male patients who were diagnosed with severe OSAS by polysomnography were evaluated for driving competence, by looking at their driving history for 2 yr, for EDS by the Epworth Sleepiness Scale (ESS) and for mood by the Self-related Depression Scale (SDS), and then underwent nasal CPAP treatment. After 2 yr of treatment, questionnaires inquiring about the patients' use of CPAP, their ESS, SDS and driving history during treatment were sent to the patients. A total of 47 patients (63%) responded to these questionnaires. Forty-six of the 47 responders had continued to use the nasal CPAP and completed the questionnaire. No

Plans are on the blackboard now for the AARC Summer Forum to be convened in Naples, FL, July 21–23, 2001. This year the Forum will run from Saturday through Monday. Look for more details this spring on the AARC web site at www.aarc.org and in *AARC Times*, the official news and feature magazine of the American Association for Respiratory Care.

traffic car accidents were observed among the 39 routine car users during treatment, while 13 of 39 patients (33%) had had car accidents before treatment. Although near-miss accidents had been reported by 32 of 39 patients (82%) before treatment, only four patients reported near-miss accidents during nasal CPAP treatment. The mean score of ESS was significantly ($p<0.01$) reduced in 46 patients after nasal CPAP. The mean score of SDS was also decreased ($p<0.01$) after nasal CPAP in 46 patients. Although 26 of 41 patients had been depressive on SDS before treatment, the mood was improved in 13 patients after nasal CPAP. These results suggest that long-term nasal CPAP treatment reduces the rate of traffic car accidents and improves the EDS and the mood in patients with OSAS.

On-Line Monitoring of Intrinsic PEEP in Ventilator-Dependent Patients—Nucci G, Mergoni M, Bricchi C, Polese G, Cobelli C, Rossi A. J Appl Physiol 2000 Sep;89(3):985-995.

Measurement of the intrinsic positive end-expiratory pressure ($PEEP_i$) is important in planning the management of ventilated patients. Here, a new recursive least squares method for on-line monitoring of $PEEP_i$ is proposed for mechanically ventilated patients. The procedure is based on the first-order model of respiratory mechanics applied to experimental measurements obtained from eight ventilator-dependent patients ventilated with four different ventilatory modes. The model $PEEP_i$ ($PEEP_{i,mod}$)was recursively constructed on an inspiration-by-inspiration basis. The results were compared with two well-established techniques to assess $PEEP_i$: end-expiratory occlusion to measure static $PEEP_i$ ($PEEP_{i,st}$ and change in airway pressure preceding the onset of inspiratory airflow to measure dynamic $PEEP_i$ ($PEEP_{i,dyn}$). $PEEP_{i,\ mod}$ was significantly correlated with both $PEEP_{i,dyn}$ ($r = 0.77$) and $PEEP_{i,st}$ ($r = 0.90$). $PEEP_{i,mod}$ (5.6 ± 3.4 cm H_2O) was systematically > $PEEP_{i,dyn}$ and $PEEP_{i,st}$ (2.7 ± 1.9 and 8.1 ± 5.5 cm H_2O, respectively), in all the models without external PEEP. Focusing on the five patients with chronic obstructive pulmonary disease, $PEEP_{i,\ mod}$ was significantly correlated with $PEEP_{i,st}$ ($r = 0.71$), whereas $PEEP_{i,dyn}$ ($r = 0.22$) was not. When PEEP was set 5 cm H_2O above $PEEP_{i,st}$, all the methods correctly estimated total PEEP, i.e., 11.8 ± 5.3, 12.5 ± 5.0, and 12.0 ± 4.7 cm H_2O for $PEEP_{i,mod}$, $PEEP_{i,st}$, and $PEEP_{i,dyn}$, respectively, and were highly correlated (0.97-0.99). We interpreted $PEEP_{i,mod}$ as the lower bound of $PEEP_{i,st}$ and concluded that our method is suitable for on-line monitoring of $PEEP_i$ in mechanically ventilated patients.

Inhaled Zanamivir for the Prevention of Influenza in Families—Hayden FG, Gubareva LV, Monto AS, Klein TC, Elliott MJ, Hammond JM, et al. N Engl J Med 2000 Nov 2;343(18):1282-1289.

Background: As prophylaxis against influenza in families, amantadine and rimantadine have had inconsistent effectiveness, partly because of the transmission of drug-resistant variants from treated index patients. We performed a double-blind, placebo-controlled study of inhaled zanamivir for the treatment and prevention of influenza in families. Methods: We enrolled families (with two to five members and at least one child who was five years of age or older) before the 1998-1999 influenza season. If an influenza-like illness developed in one member, the family was randomly assigned to receive either inhaled zanamivir or placebo. The family member with the index illness was treated with either 10 mg of inhaled zanamivir (163 subjects) or placebo (158) twice a day for 5 days, and the other family members received either 10 mg of zanamivir (414 subjects) or placebo (423) once a day as prophylaxis for 10 days. The primary end point was the proportion of families in which at least one household contact had symptomatic, laboratory-confirmed influenza. Results: The proportion of families with at least one initially healthy household contact in whom influenza developed was smaller in the zanamivir group than in the placebo group (4 percent vs. 19 percent, p <

0.001); the difference represented a 79 percent reduction in the proportion of families with at least one affected contact. Zanamivir provided protection against both influenza A and influenza B. A neuraminidase-inhibition assay and sequencing of the neuraminidase and hemagglutinin genes revealed no zanamivir-resistant variants. Among the subjects with index cases of laboratory-confirmed influenza, the median duration of symptoms was 2.5 days shorter in the zanamivir group than in the placebo group (5.0 vs 7.5 days, $p = 0.01$). Zanamivir was well tolerated. Conclusions: When combined with the treatment of index cases, prophylactic treatment of family members with once-daily inhaled zanamivir is well tolerated and prevents the development of influenza. In this study there was no evidence of the emergence of resistant influenza variants.

Ethical Decision Making and Patient Autonomy: A Comparison of Physicians and Patients in Japan and the United States—Ruhnke GW, Wilson SR, Akamatsu T, Kinoue T, Takashima Y, Goldstein MK, et al. Chest 2000 Oct;118(4):1172-1182.

BACKGROUND: Patient-centered decision making, which in the United States is typically considered to be appropriate, may not be universally endorsed, thereby harboring the potential to complicate the care of patients from other cultural backgrounds in potentially unrecognized ways. This study compares the attitudes toward ethical decision making and autonomy issues among academic and community physicians and patients of medical center outpatient clinics in Japan and the United States. METHODS: A questionnaire requesting judgments about seven clinical vignettes was distributed (in English or Japanese) to sample groups of Japanese physicians ($n = 400$) and patients ($n = 65$) as well as US physicians ($n = 120$) and patients ($n = 60$) that were selected randomly from academic institutions and community settings in Japan (Tokyo and the surrounding area) and the United States (the Stanford/Palo Alto, CA, area). Responses were obtained from 273 Japanese physicians (68%), 58 Japanese patients (89%), 98 US physicians (82%), and 55 US patients (92%). Physician and patient sample groups were compared on individual items, and composite scores were derived from subsets of items relevant to patient autonomy, family authority, and physician authority. RESULTS: A majority of both US physicians and patients, but only a minority of Japanese physicians and patients, agreed that a patient should be informed of an incurable cancer diagnosis before their family is informed and that a terminally ill patient wishing to die immediately should not be ventilated, even if both the doctor and the patient's family want the patient ventilated (Japanese physicians and patients vs US physicians and patients, $p < 0.001$). A majority of respondents in both Japanese sample groups, but only a minority in both US sample groups, agreed that a patient's family should be informed of an incurable cancer diagnosis before the patient is informed and that the family of an HIV-positive patient should be informed of this disease status despite the patient's opposition to such disclosure (Japanese physicians and patients vs US physicians and patients, $p < 0.001$). Physicians in both Japan and the United States were less likely than patients in their respective countries to agree with physician assistance in the suicide of a terminally ill patient (Japanese physicians and patients vs US physicians and patients, $p < 0.05$). Across various clinical scenarios, all four respondent groups accorded greatest authority to the patient, less to the family, and still less to the physician when the views of these persons conflicted. Japanese physicians and patients, however, relied more on family and physician authority and placed less emphasis on patient autonomy than the US physicians and patients sampled. Younger respondents placed less emphasis on family and physician authority. CONCLUSIONS: Family and physician opinions are accorded a larger role in clinical decision making by the Japanese physicians and patients sampled than by those in the United States, although both cultures place a greater emphasis on patient preferences than on the preferences of the family or physician. Our results are consistent with the view that cultural context shapes the relationship of the patient, the physician, and the patient's family in medical decision making. The results emphasize the need for clinicians to be aware of these issues that may affect patient and family responses in different clinical situations, potentially affecting patient satisfaction and compliance with therapy.

Medical and Surgical Treatment of Parapneumonic Effusions: An Evidence-Based Guideline—Colice GL, Curtis A, Deslauriers J, Heffner J, Light R, Littenberg B, et al. Chest 2000 Oct;118(4):1158-1171.

EVIDENCE: The literature review revealed 24 articles eligible for full review by the panel, 19 of which dealt with the primary management approach to PPE and 5 with a rescue approach after a previous approach had failed. Of the 19 involving the primary management approach to PPE, there were 3 randomized, controlled trials, 2 historically controlled series, and 14 case series. The number of patients included in the randomized controlled trials was small; methodologic weaknesses were found in the 19 articles describing the results of primary management approaches to PPE. The proportion and 95% CI of patients suffering each of the two relevant outcomes (death and need for a second intervention to manage the PPE) were calculated for the pooled data for each management approach from the 19 articles on the primary management approach. The pooled proportion of deaths was higher for the no drainage (6.6%), therapeutic thoracentesis (10.3%), and tube thoracostomy management approaches (8.8%) than for the fibrinolytic (4.3%), VATS (4.8%), and surgery (1.9%) approaches, but the 95% CI showed considerable overlap among all six possible primary management approaches. The pooled proportion of patients needing a second intervention to manage the PPE was also higher for the no drainage (49.2%), therapeutic thoracentesis (46.3%), and tube thoracostomy (40.3%) management approaches than the fibrinolytic (14.9%), VATS (0%), and surgery (10.7%) approaches; there was no overlap in the 95% CI between the first three and the last three management approaches, indicating a nonrandom difference. RECOMMENDATIONS: The studies identified through a careful literature review as relevant to the medical and surgical management of PPE have significant methodological limitations. Despite these limitations in the data, there did appear to be consistent and possibly clinically meaningful trends for the pooled data and the results of the randomized, controlled trials and the historically controlled series on the primary management approach to PPE. Based on these trends and consensus opinion, the panel recommends the following approach to managing PPE: In all patients with acute bacterial pneumonia, the presence of a PPE should be considered. Recommendation based on level C evidence. In patients with PPE, the estimated risk for poor outcome, using the panel recommended approach based on pleural space anatomy, pleural fluid bacteriology, and pleural fluid chemistry, should be the basis for determining whether the PPE should be drained. Recommendation based on level D evidence. Patients with category 1 or category 2 risk for poor outcome with PPE may not require drainage. Recommendation based on level D evidence. Drainage is recommended for management of category 3 or 4 PPE based on pooled data for mortality and the need for second interventions with the no drainage approach. Recommendation based on level C evidence. Based on the pooled data for mortality and the need for second interventions, therapeutic thoracentesis or tube thoracostomy alone appear to be insufficient treatment for managing most patients with category 3 or 4 PPE. Recommendation based on level C evidence. However, the panel recognizes that in the individual patient, therapeutic thoracentesis or tube thoracostomy, as planned interim steps before a subsequent drainage procedure, may result in complete resolution of the PPE. Careful evaluation of the patient for several hours is essential in these cases. If resolution occurs, no further intervention is necessary. Recommendation based on level D evidence. Fibrinolytics, VATS, and surgery are acceptable approaches for managing patients with category 3 and category 4 PPE based on cumulative data across all studies that indicate that these interventions are associated with the lowest mortality and need for second interventions. Recommendation based on level C evidence.

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Acute Respiratory Failure in the United States: Incidence and 31-Day Survival—Behrendt CE. Chest 2000 Oct;118(4):1100-1105.

Study objectives: To estimate the incidence of acute respiratory failure (ARF) in the United States and to analyze 31-day hospital mortality among a cohort of patients with ARF. Design and setting: Retrospective cohort drawn from the Nationwide Inpatient Sample of 6. 4 million discharges from 904 representative nonfederal hospitals during 1994. PATIENTS: All 61,223 patients in the sample whose discharge records indicated all of the following: acute respiratory distress or failure, mechanical ventilation, ≥ 24 h of hospitalization, and age ≥ 5 years. RESULTS: An estimated 329,766 patients discharged from nonfederal hospitals nationwide in 1994 met study criteria for ARF. The incidence of ARF was 137.1 hospitalizations per 100,000 US residents age≥ 5 years. Incidence increased nearly exponentially each decade until age 85 years. Overall, 35.9% of patients with ARF did not survive to hospital discharge. At 31 days, hospital mortality was 31.4%. According to the proportional hazards model, significant mortality hazards included age (≥ 80 years and ≥ 30 years), multiorgan system failure (MOSF), HIV, chronic liver disease, and cancer. Hospital admission for coronary artery bypass, drug overdose, or trauma other than head injury or burns was associated with a reduced mortality hazard. Interaction was present between age and MOSF, trauma, and cancer. A point system derived from the hazard model classified patients into seven groups with distinct 31-day survival probabilities ranging from 24 to 99%. CONCLUSIONS: The incidence of ARF increases markedly with age and is especially high among persons ≥ 65 years of age. Nonpulmonary hazards explain short-term (31-day) survival.

Bedside Detection of Retained Tracheobronchial Secretions in Patients Receiving Mechanical Ventilation: Is It Time for Tracheal Suctioning?—Guglielminotti J, Alzieu M, Maury E, Guidet B, Offenstadt G. Chest 2000 Oct;118(4):1095-1099.

OBJECTIVE: To identify parameters that indicate retained secretions and the need for tracheal suctioning (TS) in patients receiving mechanical ventilation (MV). DESIGN: Prospective observational study. SETTING: A 14-bed medical ICU in a 946-bed university hospital. PATIENTS: Sixty-six consecutive patients receiving MV. INTERVENTIONS: Two successive tracheal suctions, TS1 and TS2, performed at a 2-h interval as usual patient care. Retained secretions were considered significant if the volume of secretions removed by TS2 was > 0.5 mL. Measurements and results: Variations between TS1 and TS2 of pulse oximetric saturation (S_{pO_2}), peak inspiratory pressure (P_{peak}), tidal volume (V_T), and Ramsay score were compared between patients with TS2 ≤ 0.5 mL (group 1; n = 27) and patients with TS2 > 0.5 mL (group 2; n = 39). The presence of a sawtooth pattern on flow-volume loop displayed on the monitor screen of the ventilator and of respiratory sounds heard over the trachea before TS2 were compared between the two groups. Variations of P_{peak}, V_T, S_{pO_2}, and Ramsay score between TS1 and TS2 did not differ between the two groups. However, group 2 had a sawtooth pattern (82% vs 29.6%; p = 0.0001) and respiratory sounds (66.6% vs 25.9%; p 0.001) more frequently than group 1 before TS2. For the sawtooth pattern, the likelihood ratio (LR) of a positive test was 2.70 and the LR of a negative test was 0.25, while for respiratory sounds it was 2.50 and 0.45, respectively. When the presence of a sawtooth pattern and of respiratory sounds was combined, the LR of a positive test rose to 14.7 and the LR of a negative test was 0.42. CONCLUSIONS: A sawtooth pattern and/or respiratory sounds over the trachea are good indicators of retained secretions in patients receiving MV and may indicate the need for TS. Conversely, the absence of a sawtooth pattern may rule out retained secretions.

Targeting Aerosol Deposition in Patients with Cystic Fibrosis: Effects of Alterations in Particle Size and Inspiratory Flow Rate—Laube BL, Jashnani R, Dalby RN, Zeitlin PL. Chest 2000 Oct;118(4): 1069-1076.

Study objective: To determine if aerosolized medications can be targeted to deposit in the smaller, peripheral airways or the larger, central airways of adult cystic fibrosis (CF) patients by varying particle size and inspiratory flow rate. DESIGN: Randomized clinical trial. SETTING: Outpatient research laboratory. PATIENTS: Nine adult patients with CF. INTERVENTIONS: Patients inhaled an aerosol comprised of 3.68 ± 0.04 μm saline solution droplets (two visits) or 1.01 ± 0.2 μm saline solution droplets (two visits) for 30 s, starting from functional residual capacity and breathing at a slow or faster inspiratory flow rate. On all visits, the saline solution was admixed with the radioisotope ^{99m}Tc. Immediately after inhalation, a gamma camera recorded the deposition pattern of the radioaerosol in the lungs. Deposition images were analyzed in terms of the inner:outer zone (I:O) ratio, a measure of deposition in an inner zone (large, central airways) vs an outer zone (small airways and alveoli). Measurements and results: For the 3.68-μm aerosol, I:O ratios averaged 2.29 ± 1.45 and 2.54 ± 1.48 ($p > 0.05$), indicating that aerosol distribution within the lungs was unchanged while breathing at 12 ± 2 L/min vs 31 ± 5 L/min, respectively. For the 1.01-μm aerosol, I:O ratios averaged 2.09 ± 0.96 and 3.19 ± 1.95 ($p < 0.05$), indicating that deposition was predominantly in the smaller airways while breathing at 18 ± 5 L/min and in the larger airways while breathing at 38 ± 8 L/min, respectively. CONCLUSIONS: These results suggest that the targeted delivery of an aerosol to the smaller, peripheral airways or the larger, central airways of adult CF patients may be achieved by generating an aerosol comprised of approximately 1.0-μm particles and inspiring from functional residual capacity at approximately 18 L/min and approximately 38 L/min, respectively.

Vertebral Fracture Risk with Long-Term Corticosteroid Therapy: Prevalence and Relation to Age, Bone Density, and Corticosteroid Use—Naganathan V, Jones G, Nash P, Nicholson G, Eisman J, Sambrook PN. Arch Intern Med 2000 Oct 23;160(19):2917-2922.

BACKGROUND: Few data are available regarding vertebral fracture risk in patients treated with oral corticosteroids. The aim of this study was to determine the prevalence and the role of risk factors such as age, bone mineral density (BMD), and corticosteroid use for vertebral deformity in patients receiving long-term corticosteroid therapy. METHODS: Thoracolumbar x-ray films, BMD, and details on corticosteroid use were obtained on 229 consecutive patients treated with long-term corticosteroid regimens (≥ 6 months of prednisone, ≥ 5mg/d or equivalent) seen at 4 referral centers. Comparisons were made with a population control group of 286 male and female controls not taking corticosteroids (aged ≥ 60 years). RESULTS: Sixty-five patients (28%) had at least 1 vertebral deformity and 25 (11%) had 2 or more vertebral deformities. Older age, independent of BMD, was a significant risk factor for deformity. Patients aged 70 to 79 years had a 5-fold increased risk of deformity compared with patients younger than 60 years (odds ratio, 5.13; 95% confidence interval, 2. 03-13.0). Compared with the population controls, the prevalence of deformities increased to a greater extent with each decade of age in the corticosteroid group ($p = 0.005$). Mean lumbar spine and femoral neck BMD Z scores were lower in the steroid-treated patients with deformities compared with the nonsteroid control group with deformities. When the effects of age, sex, body mass index, and duration of corticosteroid use were adjusted for in logistic regression analysis, low BMD was a modest predictor of deformity (for a 1-SD decrease in lumbar spine BMD: odds ratio, 1.31; 95% confidence interval, 1.02-1.68) and for a 1-SD decrease in femoral neck BMD: odds ratio, 1.77; 95% confidence interval, 1.07-2.94). CONCLUSIONS: The combination of increasing age and corticosteroid use is associated with a marked increase in the risk of vertebral deformity. Elderly patients commencing long-term corticosteroid therapy should be considered for antiosteoporotic therapy independently of their BMD.

Early Exposure to House-Dust Mite and Cat Allergens and Development of Childhood Asthma: A Cohort Study—Lau S, Illi S, Sommerfeld C, Niggemann B, Bergmann R, von Mutius E, and the Multicentre Allergy Study Group. Lancet 2000 Oct 21;356(9239):1392-1397.

BACKGROUND: In a prospective birth-cohort study, we assessed the relevance of mite and cat allergen exposure for the development of childhood asthma up to age 7 years. METHODS: Of 1314 newborn infants enrolled in five German cities in 1990, follow-up data at age 7 years were available for 939 children. Assessments included repeated measurement of specific IgE to food and inhalant allergens, measurement of indoor allergen exposure at 6 months, 18 months, and 3 years of age, and yearly interviews by a paediatrician. At age 7 years, pulmonary function was tested and bronchial hyper-responsiveness was measured in 645 children. FINDINGS: At age 7, the prevalence of wheezing in the past 12 months was 10.0% (94 of 938), and 6.1% (57 of 939) parents reported a doctor's diagnosis of asthma in their children. Sensitisation to indoor allergens was associated with asthma, wheeze, and increased bronchial responsiveness. However, no relation between early indoor allergen exposure and the prevalence of asthma, wheeze, and bronchial hyper-responsiveness was seen. INTERPRETATION: Our data do not support the hypothesis that exposure to environmental allergens causes asthma in childhood, but rather that the induction of specific IgE responses and the development of childhood asthma are determined by independent factors.

Relation between Respiratory Changes in Arterial Pulse Pressure and Fluid Responsiveness in Septic Patients with Acute Circulatory Failure—Michard F, Boussat S, Chemla D, Anguel N, Mercat A, Lecarpentier Y, et al. Am J Respir Crit Care Med 2000 Jul;162(1):134-138.

In mechanically ventilated patients with acute circulatory failure related to sepsis, we investigated whether the respiratory changes in arterial pressure could be related to the effects of volume expansion (VE) on cardiac index (CI). Forty patients instrumented with indwelling systemic and pulmonary artery catheters were studied before and after VE. Maximal and minimal values of pulse pressure (Pp_{max} and Pp_{min}) and systolic pressure (Ps_{max} and Ps_{min}) were determined over one respiratory cycle. The respiratory changes in pulse pressure (ΔPp) were calculated as the difference between Pp_{max} and Pp_{min} divided by the mean of the two values and were expressed as a percentage. The respiratory changes in systolic pressure (ΔPs) were calculated using a similar formula. The VE-induced increase in CI was ≥ 15% in 16 patients (responders) and < 15% in 24 patients (nonresponders). Before VE, ΔPp (24 ± 9 versus 7 ± 3%, $p < 0.001$) and ΔPs (15 ± 5 versus 6 ± 3%, $p < 0.001$) were higher in responders than in nonresponders. Receiver operating characteristic (ROC) curves analysis showed that ΔPp was a more accurate indicator of fluid responsiveness than ΔPs. Before VE, a ΔPp value of 13% allowed discrimination between responders and nonresponders with a sensitivity of 94% and a specificity of 96%. VE induced changes in CI closely correlated with ΔPp before volume expansion ($r^2 = 0.85$, $p < 0.001$). VE decreased ΔPp from 14 ± 10 to 7 ± 5% ($p < 0.001$) and VE-induced changes in ΔPp correlated with VE-induced changes in CI ($r^2 = 0.72$, $p < 0.001$). It was concluded that in mechanically ventilated patients with acute circulatory failure related to sepsis, analysis of ΔPp is a simple method for predicting and assessing the hemodynamic effects of VE, and that ΔPp is a more reliable indicator of fluid responsiveness than ΔPs.

Hemodynamic Tolerance of Intermittent Hemodialysis in Critically Ill Patients: Usefulness of Practice Guidelines—Schortgen F, Soubrier N, Delclaux C, Thuong M, Girou E, Brun-Buisson C, et al. Am J Respir Crit Care Med 2000 Jul;162(1):197-202.

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PROGRAM GUIDE

PROGRAM #1

Taking the Mystery Out of Weaning the Pediatric Patient from the Ventilator

Peter Betit, BS, RRT, FAARC, and Richard D. Branson, BA, RRT, FAARC

Learn when to begin the process and how to recognize critical events in weaning a pediatric patient. Also teaches the physiological differences between the adult and pediatric patient and why weaning of the pediatric patient is different. The presentation confronts participants with options in providing assisted ventilation and the correct selection of options that expedite weaning.

Live Videoconference -
March 13, 11:30 a.m. - 1:00 p.m. Central Time
Teleconference with Videotape -
April 10, 11:30 a.m. - 12:00 Noon Central Time

PROGRAM #2

Pulmonary Rehabilitation: Standard Care for Chronic Lung Disease Patients

Trina Limberg, BS, RRT, and Thomas J. Kallstrom, RRT, FAARC

Presentation details when to refer a patient for pulmonary rehabilitation and the four elements necessary for the successful operation of a rehabilitation service. Details how to prepare a treatment plan during assessment and how to modify it based on subsequent evaluations as well as how to incorporate rehabilitation techniques into routine bedside therapy sessions.

Live Videoconference -
March 27,11:30 a.m. - 1:00 p.m. Central Time
Teleconference with Videotape -
April 17, 11:30 a.m. - 12:00 Noon Central Time

PROGRAM #3

Noninvasive Ventilation: The Latest Word

Dean R. Hess, PhD, RRT, FAARC, and Richard D. Branson, BA, RRT, FAARC

Learn how to avoid intubation in the acutely ill patient through identification of patients most likely to benefit from noninvasive ventilation. Learn selection and proper fit of full masks or nasal masks and how to select the proper ventilator based on the patient's condition and desired outcomes. Also learn when to make adjustments to achieve the goals of unloading respiratory muscles and achieving good patient/ventilator synchrony.

Live Videoconference -
April 24, 11:30 a.m. - 1:00 p.m. Central Time
Teleconference with Videotape -
May 29, 11:30 a.m. - 12:00 Noon Central Time

PROGRAM #4

Education of the Patient with Asthma

Tracey Mitchell, RRT, RPFT, and Thomas J. Kallstrom, RRT, FAARC

This program teaches how to ensure that patients understand the disease process of asthma and their care plan for effective disease management. And, it details what patient education materials are available, their content, where to find them, and the best methods of presentation, including new terminologies, analogies, and techniques.

Live Videoconference -
May 22, 11:30 a.m. - 1:00 p.m. Central Time
Teleconference with Videotape -
June 19, 11:30 a.m. - 12:00 Noon Central Time

PROGRAM #5

ARDS: The Disease and Its Management

Leonard D. Hudson, MD, and David J. Pierson, MD, FAARC

Presents the four diagnostic criteria for ARDS and the six clinical risk factors that place patients at increased likelihood for developing ARDS. The program will teach viewers how to understand the implications of the lower and upper inflection points on the pressure-volume curve of the respiratory system in ARDS patients; and instruct them in the calculation of estimated required tidal volume.

Live Videoconference -
June 26, 11:30 a.m. - 1:00 p.m. Central Time
Teleconference with Videotape -
July 17, 11:30 a.m. - 12:00 Noon Central Time

PROGRAM #6

New Respiratory Drugs: What, When, and How?

Joseph L. Rau, PhD, RRT, FAARC, and Patrick J. Dunne, MEd, RRT, FAARC

Introduces participants to new formulations such as racemic drug mixtures and single isomers and their effective duration and how they lead to lower costs with improved patient responses. Viewers will learn the use of improved anticholinergics in the treatment of asthma patients and learn the uses and effects of inhaled anti-infective agents.

Live Videoconference -
Aug.14, 11:30 a.m. - 1:00 p.m. Central Time
Teleconference with Videotape -
Sept.11, 11:30 a.m. - 12:00 Noon Central Time

PROGRAM #7

Invasive Ventilation: The Latest Word

Richard Kallet, MS, RRT and Richard D. Branson, BA, RRT FAARC

Learn how proper ventilator management can preclude inflicting harm on the patient and why it is essential for the clinician to understand the function and mechanics of newer mechanical ventilators. Also learn how reducing the patient's work of breathing is essential in reducing the additional load on ventilatory musculature, and why reinflating lungs and enhancing the functional area of the lung demands extraordinary means.

Live Videoconference -
Sept. 25, 11:30 a.m. - 1:00 p.m. Central Time
Teleconference with Videotape -
Oct. 16, 11:30 a.m. - 12:00 Noon Central Time

PROGRAM #8

Test Your Lungs, Know Your Numbers, Prevent Emphysema

Thomas L. Petty, MD, FAARC and David J. Pierson, MD, FAARC

Reviews the classic signs of COPD with an emphasis on emphysema and a discussion on the measures used to relieve symptoms and slow disease progression. Covers the importance of pulmonary function tests to determine VC, FFC, and FEV_1; and why getting patients to know their numbers is the key to early diagnosis and successful treatment.

Live Videoconference -
Oct. 23, 11:30 a.m. - 1:00 p.m. Central Time
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Nov. 20, 11:30 a.m. - 12:00 Noon Central Time

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Poor hemodynamic tolerance of intermittent hemodialysis (IHD) is a common problem for patients in an intensive care unit (ICU). New dialysis strategies have been adapted to chronic hemodialysis patients with cardiovascular insufficiency. To improve hemodynamic tolerance of IHD, specific guidelines were progressively implemented into practice through the year 1996 in our 26-bed medical ICU. To evaluate the efficiency of these guidelines we retrospectively compared all IHD performed during the years before (1995) and after (1997) implementation of these recommendations. Forty-five patients underwent 248 IHD sessions in 1995 and 76 patients underwent 289 IHD sessions in 1997. The two populations were similar for age, sex, chronic hemodialysis (26% versus 17%), and secondary acute renal failure. In 1997, patients were more severely ill with a higher SAPS II (50 ± 17 versus 59 ± 24; $p = 0.036$), and more patients required epinephrine or norepinephrine infusion before dialysis sessions (16% versus 34%; $p < 0.0001$). The compliance to guidelines was high, inducing a significant change in IHD modalities. As a result, hemodynamic tolerance was significantly better in 1997, with less systolic blood pressure drop at onset (33% versus 21%, $p = 0.\ 002$) and during the sessions (68% versus 56%, $p = 0.002$). IHD with hypotensive episode or need for therapeutic interventions were less frequent in 1997 (71% versus 61%, $p = 0.015$). The ICU mortality was similar (53.3% in 1995 versus 47.3% in 1997; $p = 0.52$) but death rate in 1997, but not in 1995, was significantly less than predicted from SAPS II (47.3% versus 65.6%; $p = 0.02$). Length of ICU stay was also reduced for survivors in 1997 ($p = 0.04$). Implementation of practice guidelines for intermittent hemodialysis in ICU patients lessens hemodynamic instability and may improve outcome.

The Prevalence of Gastroesophageal Reflux in Asthma Patients without Reflux Symptoms—Harding SM, Guzzo MR, Richter JE. Am J Respir Crit Care Med 2000 Jul;162(1):34-39.

Gastroesophageal reflux is a potential trigger of asthma that may be clinically silent. This study examines the prevalence of gastroesophageal reflux in asthma patients without reflux symptoms. This prospective cohort study evaluated 26 patients with stable asthma without reflux symptoms using esophageal manometry and 24-h esophageal pH testing. Gastroesophageal reflux was considered present if esophageal acid contact times were abnormal. Demographic variables were analyzed to determine if they predicted the presence of gastroesophageal reflux. Asthma patients with asymptomatic gastroesophageal reflux were compared with 30 age-matched asthma patients with symptomatic gastroesophageal reflux. The prevalence of abnormal 24-h esophageal pH tests in asthma patients without reflux symptoms was 62% (16 of 26). Demographic variables did not predict abnormal 24-h esophageal pH tests in asthma patients with asymptomatic gastroesophageal reflux. Asthma patients with asymptomatic gastroesophageal reflux had higher amounts of proximal esophageal acid exposure ($p < 0.05$) compared with asthma patients with symptomatic gastroesophageal reflux. Because demographic variables do not predict abnormal 24-h esophageal pH tests in asthma patients without reflux symptoms, 24-h esophageal pH testing is required. This study suggests that gastroesophageal reflux is present in asthma patients, even in the absence of esophageal symptoms.

Differences in Incidence of Reported Asthma Related to Age in Men and Women: A Retrospective Analysis of the Data of the European Respiratory Health Survey—de Marco R, Locatelli F, Sunyer J, Burney P. Am J Respir Crit Care Med 2000 Jul;162(1):68-74.

Sex differences in asthma prevalence and morbidity, assessed with different methods in different populations, have raised several hypotheses about the different susceptibility to asthma in men and women. However, information on the incidence of asthma by age and sex is limited. The aim of this study was to estimate the age- and sex-specific incidence of asthma from birth to 44 yr of age in men and women across several countries, and to evaluate the main factors influencing asthma incidence in young adults. The data of the European Community Respiratory Health Survey, an international, cross sectional, population-based survey, which were collected in 16 countries from 1991 to 1993 according to a common protocol, and which pertained to 18,659 subjects, were analyzed retrospectively, using the reported age of the first attack as the onset of asthma. During childhood, girls had a significantly lower risk of developing asthma than did boys (relative risk [RR]: 0.74 and 0.56 in the 0- to 5-yr and 5- to 10-yr age classes, respectively). Around puberty, the risk was almost equal in the two sexes (RR = 0.84; 95% confidence interval [CI]: 0.65 to 1.10 in the 10- to 15-yr age class). After puberty, the risk in women was always significantly higher than that in men (RR: 1.38 to 5.91). This pattern was consistent in all of the 16 countries studied, and was not influenced by recall or cohort effects. When the effects of airway caliber and smoking were studied with a case control design, the results showed that women's greater susceptibility to asthma in early adulthood was at least partly explained by their smaller airway caliber (the OR decreased from 2. 04 [95% CI: 1.32 to 3.15] to 1.47 [95% CI: 0.89 to 2.44] after controlling for height adjusted FEV_1); while smoking did not increase the risk. This analysis strongly confirms that the incidence of asthma shows a sex reversal during puberty, and suggests that airway caliber, in addition to hormonal factors, could play an important role in explaining the different patterns of asthma incidence in men and women.

Adverse Ventilatory Strategy Causes Pulmonary-to-Systemic Translocation of Endotoxin—Murphy DB, Cregg N, Tremblay L, Engelberts D, Laffey JG, Slutsky AS, et al. Am J Respir Crit Care Med 2000 Jul;162(1):27-33.

Accumulating evidence strongly suggests that ventilatory strategy has an important impact on development of lung injury and patient outcome. Adverse ventilatory strategies have been shown to cause release of pulmonary derived cytokines and may permit bacterial translocation from the lung to the systemic circulation. Because endotoxin is a potent and clinically important stimulant of cytokine-mediated systemic inflammatory responses that can lead to multiorgan failure, we investigated the effects of ventilatory strategy on lung-to-systemic-translocation of endotoxin. We studied the effects of protective (tidal volume-[V_T] $5\ mL \cdot kg^{-1}$, positive end-expiratory pressure [PEEP] 10 to 12.5 cm H_2O) versus nonprotective (V_T $12\ mL \cdot kg^{-1}$, PEEP zero) ventilatory strategy on translocation of endotracheally instilled endotoxin. Anesthetized New Zealand White rabbits were subjected to saline lung lavage, and 32 were randomized to one of four groups: PS (protective ventilation + instilled saline); PE (protective ventilation + instilled endotoxin); NS (nonprotective ventilation + instilled saline); NE (nonprotective ventilation + instilled endotoxin), and ventilated for 3 h. Plasma endotoxin levels increased significantly in the NE group, and remained low and unchanged in the other groups. Peak levels of plasma tumor necrosis factor-alpha (TNF-α) were higher in NE versus other groups. P_{aO_2} and mean arterial pressure ($\bar{P}a$) were lowest, and requirement for pressor and bicarbonate support greatest, in the NE group. Finally, plasma endotoxin levels were significantly greater in eventual nonsurvivors than survivors. These data provide convincing evidence for pulmonary translocation of lung-derived endotoxin. This translocation depends on ventilatory strategy, and suggests a pathophysiologic link between ventilatory strategy and outcome.

Respiratory Disturbance Index: An Independent Predictor of Mortality in Coronary Artery Disease—Peker Y, Hedner J, Kraiczi H, Loth S. Am J Respir Crit Care Med 2000 Jul;162(1):81-86.

Cardiovascular mortality was prospectively investigated in consecutive coronary artery disease (CAD) patients with versus without obstructive sleep apnea (OSA) during a follow up period of 5 yr. An overnight sleep/ventilatory study was performed in patients requiring intensive care ($n = 62$, mean age 67.6 ± 10.4 yr, range 44 to 86) during a stable

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Asthma Drugs and Medications: What's Right and What's Wrong
Reviews the pharmacology section of the revised NAEPP Guidelines. Additionally, the potential hazards and undesirable side effects of commonly prescribed medications will be discussed, with alternative treatment regimens suggested. Featuring William Luskin, MD, and David J. Pierson, MD. 80-min. videotape.
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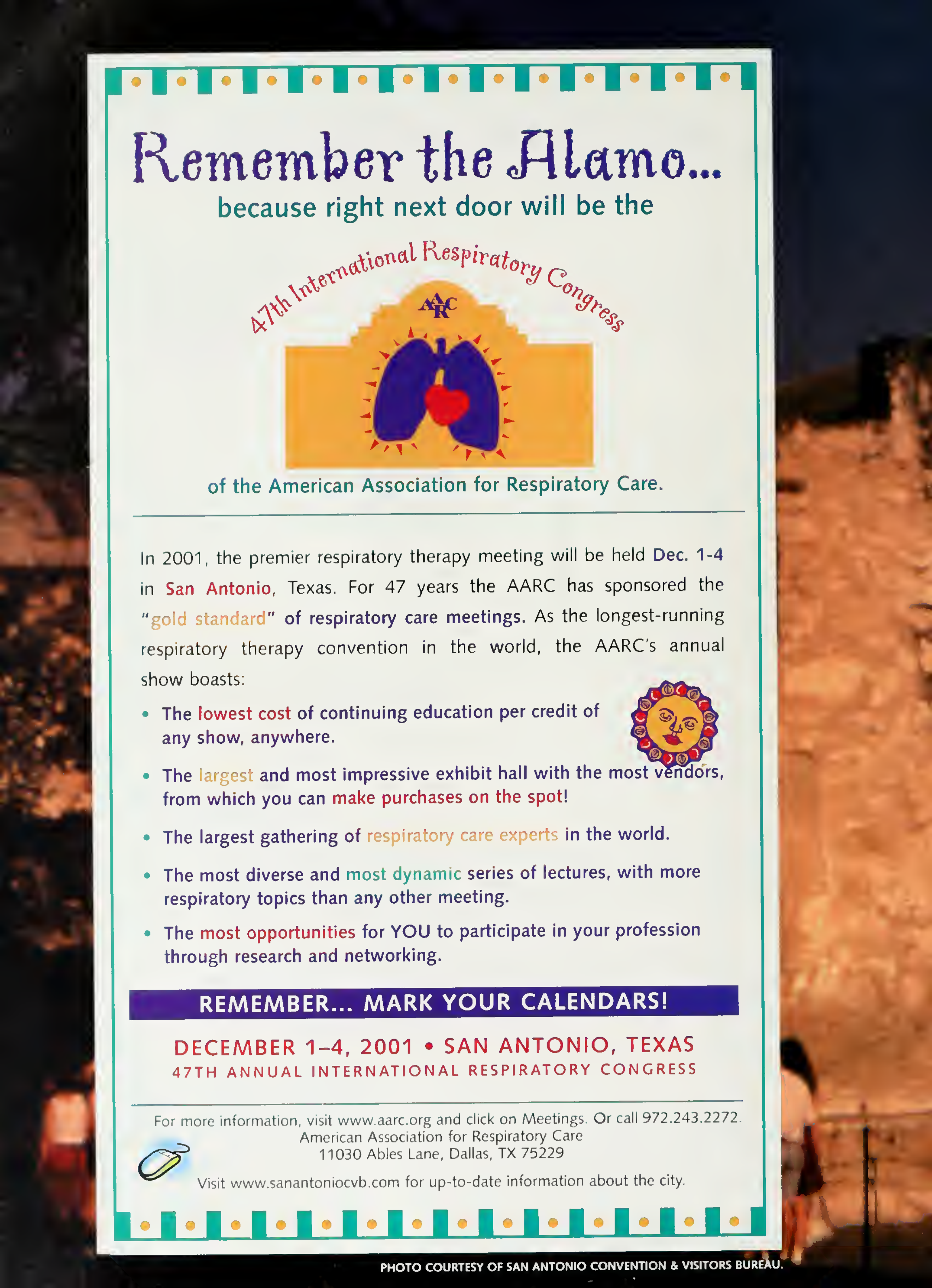

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condition (New York Heart Association [NYHA] functional class I-II) 4 to 21 mo after discharge from the hospital. OSA, defined as a respiratory disturbance index (RDI) of 10/h or more was found in 19 patients (mean RDI 17.5 ± 8.3). Three OSA subjects who were successfully treated with continuous positive airway pressure (CPAP) during the observation period were excluded from the final analysis. There was no statistically significant difference (Fisher two-tailed exact test) between the OSA and non-OSA patient groups in terms of number of elderly subjects (age ≥ 65 yr), gender, obesity (body mass index [BMI] ≥ 30 kg/m^2), smoking history, presence of hypertension, diabetes mellitus, hypercholesterolemia, or history of myocardial infarction at the study start. During the follow-up period, cardiovascular death occurred in six of 16 OSA patients (37.5%) compared with 4 (9.3%) in the non-OSA group ($p = 0.018$). The univariate predictors of cardiovascular mortality were RDI ($p = 0.007$), OSA ($p = 0.014$), age at baseline ($p = 0.028$), hypertension at baseline ($p = 0.036$), history of never-smoking ($p = 0.031$), and digoxin treatment during the follow-up period ($p = 0.013$). In a Cox multiple conditional regression model, RDI remained as an independent predictor of cardiovascular mortality (exp $\beta = 1.13$, 95% confidence interval [CI] 1.05 to 1.21, two-sided $p < 0.001$). We conclude that untreated OSA is associated with-an increased risk of cardiovascular mortality in patients with CAD. Furthermore, it appears appropriate that RDI is taken into consideration when evaluating secondary prevention models in CAD.

Comparison of Esophageal Pressure with Pulse Transit Time As a Measure of Respiratory Effort for Scoring Obstructive Nonapneic Respiratory Events—Argod J, Pepin JL, Smith RP, Levy P. Am J Respir Crit Care Med 2000 Jul;162(1):87-93.

Obstructive nonapneic respiratory events (ONAREs) are much more difficult to detect and classify than apneas unless sensitive measures of respiratory effort and airflow are employed. The aim of this study was to compare two measures of respiratory effort, esophageal pressure monitoring (Pes) and pulse transit time (PTT), for scoring of ONAREs by visual analysis. Nine men (age 49 ± 10 yr) with mild to moderate sleep apnea syndrome (AHI of 25.1 ± 10.8/h) were studied and 340 ONAREs (hypopneas and upper airway resistance episodes) were randomly selected for scoring by two experienced observers. Each observer blindly scored each ONARE twice (once with Pes and once with PTT) with a concurrent pneumotachography trace available for airflow quantification. This permitted the respiratory events scored with PTT to be compared with those scored with Pes, and in addition interobserver variability could be assessed for each signal. Even though standard criteria were used for scoring, there was significant interobserver variability for both Pes (29.7%) and PTT (37.1%). Taking those events for which there was agreement between the observers, PTT had a sensitivity of 79.9% and a positive predictive value of 91.2% (using Pes as the gold standard). In those ONAREs for which there was agreement between the two observers there was a larger percentage reduction in airflow compared to ONAREs that did not concur (51 versus 30.3%, $p < 0.001$), a larger increase in respiratory effort as assessed by PTT (slope of PTT: 23.1 versus 14. 3 arbitrary units, $p < 0.01$), and a higher incidence in autonomic microarousals detected with PTT (90 versus 45% of ONAREs, $p < 0.006$). Subtle respiratory events are more difficult to detect than apneas or frank hypopneas. When comparing PTT with esophageal pressure in detecting those events the sensitivity of PTT is good but limited when the reduction in airflow, the increase in respiratory effort, or the arousal reaction is the less clear. However, PTT appears to be a good noninvasive alternative to Pes in the detection of nonapneic obstructive respiratory events, and its ability to detect autonomic arousal gives this physiological signal added clinical usefulness.

Accuracy of an Unattended Home CPAP Titration in the Treatment of Obstructive-Sleep Apnea—Series F. Am J Respir Crit Care Med 2000 Jul;162(1):94-97.

Treatment of sleep apnea-hypopnea syndrome (SAHS) by fixed continuous positive airway pressure (CPAP) requires an in-laboratory titration procedure to determine the effective pressure level (Peff). We recently reported that one auto-CPAP machine can be used without titration study allowing Peff determination. The aim of this study was to evaluate the accuracy of an auto-CPAP trial at home. A 1- or 2-wk automatic CPAP trial was done at home in 40 patients by estimating the reference pressure (Pref) to be set and a Pref + 3 cm H_2O/4 cm H_2O pressure interval. Peff was then determined according to the percentage of CPAP time that was spent ≤ Pref. This Peff value was set on a fixed CPAP machine for two additional weeks and a control sleep study was done. The pressure setting on fixed CPAP had to be increased by 1 ± 1 cm H_2O (mean ± SD) above estimated Pref. Sleep improved with fixed CPAP, with a normalization of the apnea + hypopnea index (AHI) in 38 of 40 and resumption of diurnal hypersomnolence. CPAP compliance remained excellent (CPAP use: 6.1 ± 1.7 h/night) after 6.5 ± 2.8 mo of CPAP treatment. These results indicate that auto-CPAP therapy represents a new useful and accurate way to identify conventional CPAP setting outside hospital and sleep laboratories.

Withdrawal of Chronic Systemic Corticosteroids in Patients with COPD: A Randomized Trial—Rice KL, Rubins JB, Lebahn F, Parenti CM, Duane PG, Kuskowski M, et al. Am J Respir Crit Care Med 2000 Jul;162(1):174-178.

The benefits of chronic systemic corticosteroids for patients with chronic obstructive pulmonary disease (COPD) are not well established. To determine whether chronic corticosteroid treatment can be safely withdrawn in "steroid-dependent" COPD patients, we performed a double-blind, placebo-controlled study of 38 patients with steroid-dependent COPD. Patients were randomly assigned to receive their usual maintenance prednisone dose for 6 mo (continuous group) or to be withdrawn from prednisone at a rate of 5 mg per week (demand group). The number of COPD exacerbations per patient (primary outcome) was 2.5 ± 2.7 (mean ± SD) in the continuous group and 2.7 ± 2.5 in the demand group ($p = 0.60$, 95% confidence interval for the difference: -1.1 to 1.7). Spirometric results, dyspnea, and health-related quality of life did not differ significantly in the two groups. The average daily corticosteroid dose was 10.7 ± 5.2 mg in the continuous group and 6.3 ± 6.4 mg in the demand group ($p = 0.003$). Weight decreased in the demand group by 4.8 ± 2.0 kg, compared with an increase in the continuous group of 0.5 ± 3.5 kg ($p = 0.007$). Discontinuation of chronic systemic corticosteroid treatment in steroid-dependent COPD patients did not cause a significant increase in COPD exacerbations, but did reduce total systemic corticosteroid use and body weight. Larger studies may be warranted to establish the relative risks and benefits of-chronic corticosteroid treatment of patients with COPD.

Factor Analysis of Changes in Dyspnea and Lung Function Parameters after Bronchodilation in Chronic Obstructive Pulmonary Disease—Taube C, Lehnigk B, Paasch K, Kirsten DK, Jorres RA, Magnussen H. Am J Respir Crit Care Med 2000 Jul;162(1):216-220.

Expiratory airway collapse is a characteristic feature in patients with chronic obstructive pulmonary disease (COPD). We hypothesized that this collapse might mask the effects of bronchodilators during forced expiration but not during forced inspiration, and that accordingly, the improvement in forced inspiration and not that in forced expiration with bronchodilator therapy would be related to changes in the perception of dyspnea. In order to investigate this, we conducted lung function measurements, including measurements of forced inspiration and expiration

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before and 30 min after inhalation of 400 microg salbutamol, in 61 patients with COPD (mean FEV$_1$: 38. 3 L; range: 12.9 to 79.5% predicted). The change in dyspnea from baseline was assessed with a standard visual analogue scale (VAS) ranging from -100 to +100. To delineate the relationship between parameters, we used the statistical procedure of factor analysis. Salbutamol induced an improvement of 0.16 ± 0.02 L (mean ± SD) in FEV$_1$, 0.36 ± 0.04 L in forced inspiratory volume in one second (FEV$_1$), 0.30 ± 0.04 L in inspiratory capacity (IC), and -0.34 ± 0.07 L in intrathoracic gas volume; the mean VAS score was 36.4 ± 3.2. Factor analysis demonstrated that the reduction in dyspnea at rest was primarily associated with changes in parameters describing forced inspiration and not with those of forced expiration or lung hyperinflation, including IC. Our data indicate that in patients with COPD, the reduction in dyspnea after inhalation of a β_2-adrenoreceptor agonist is closely correlated with the change in parameters of forced inspiration, and particularly FIV$_1$, but not with changes in parameters of forced expiration or lung hyperinflation.

A Prospective, Multicenter Study of the Epidemiology, Management, and Outcome of Severe Acute Renal Failure in a "Closed" ICU System—Cole L, Bellomo R, Silvester W, Reeves JH, for the Victorian Severe Acute Renal Failure Study Group. Am J Respir Crit Care Med 2000 Jul;162(1):191-196.

The safety and effectiveness of "closed" intensive care units (ICUs) are highly controversial. The epidemiology and outcome of acute renal failure (ARF) requiring replacement therapy (severe ARF) within a "closed" ICU system are unknown. Accordingly, we performed a prospective 3-mo multicenter observational study of all Nephrology Units and ICUs in the State of Victoria (all "closed" ICUs), Australia, and focused on the epidemiology, treatment, and outcome of patients with severe ARF. We collected demographic, clinical, and outcome data using standardized case report forms. Nineteen ward patients and 116 adult ICU patients had severe ARF (13.4 cases/100,000 adults/yr). Among the ICU patients with severe ARF, 37 had impaired baseline renal function, 91 needed ventilation, and 95 needed vasoactive drugs. Intensivists controlled patient care in all cases. Continuous renal replacement therapy (CRRT) was used in 111 of the ICU patients. Nephrological opinion was sought in only 30 cases. Predicted mortality was 59.6%. Actual mortality was 49.2%. Only 11 ICU survivors were dialysis dependent at hospital discharge. In the state of Victoria, Australia, intensivists manage severe ARF within a "closed" ICU system. Renal replacement is typically continuous and outcomes compare favorably with those predicted by illness severity scores. Our findings support the safety and efficacy of a "closed" ICU model of care.

Noninvasive versus Invasive Microbial Investigation in Ventilator-Associated Pneumonia: Evaluation of Outcome—Ruiz M, Torres A, Ewig S, Marcos MA, Alcon A, Lledo R, et al. Am J Respir Crit Care Med 2000 Jul;162(1):119-125.

Noninvasive and invasive diagnostic techniques have been shown to achieve comparable performances in the evaluation of suspected ventilator-associated pneumonia (VAP). We studied the impact of both approaches on outcome in a prospective, open, and randomized study in three intensive care units (ICUs) of a 1,000-bed tertiary care university hospital. Patients with suspected VAP were randomly assigned to noninvasive (Group 1) versus invasive (Group 2) investigation (tracheobronchial aspirates [TBAS] versus bronchoscopically-retrieved protected specimen brush [PSB] and bronchoalveolar lavage [BAL].Samples were cultured quantitatively, and BAL fluid (BALF) was examined for intracellular organisms (ICO) additionally. Initial empiric antimicrobial treatment was administered following the guidelines of the American Thoracic Society (ATS) and adjusted according to culture results (and ICO counts in Group 2). Outcome variables included length of ICU stay and mechanical ventilation as well as mortality. Overall, 76 patients (39 noninvasive, 37 invasive) were investigated. VAP was microbiologically confirmed in 23 of 39 (59%) and 23 of 37 (62%) ($p = 0.78$). There were no differences with regard to the frequencies of community-acquired and potentially drug-resistant microorganisms (PDRM). Antimicrobial treatment was changed in seven patients (18%) of Group 1 and 10 patients (27%) of Group 2 because of etiologic findings (including five of 17 with ICO = 2% (p = not significant [NS]). Length of ICU stay and mechanical ventilation were also not significantly different in both groups. Crude 30-d mortality was 31 of 76 (41%), and 18 of 39 (46%) in Group 1 and 14 of 37 (38%) in Group 2 ($p = 0.46$). Adjusted mortality was 16% versus 11% ($p = 0.53$), and mortality of microbiologically confirmed pneumonia 10 of 23 (44%) in both groups ($p = 1.0$). We conclude that the outcome of VAP was not influenced by the techniques used for microbial investigation.

Permissive Hypercapnia Impairs Pulmonary Gas Exchange in the Acute Respiratory Distress Syndrome—Feihl F, Eckert P, Brimioulle S, Jacobs O, Schaller MD, Melot C, Naeije R. Am J Respir Crit Care Med 2000 Jul;162(1):209-215.

Current recommendations for mechanical ventilation in the acute respiratorydistress syndrome (ARDS) include the use of small tidal volumes (V_T), even at the cost of respiratory acidosis. We evaluated the effects of this permissive hypercapnia on pulmonary gas exchange with the multiple inert gas elimination technique (MIGET) in eight patients with ARDS. After making baseline measurements, we induced permissive hypercapnia by reducing V_T from 10 ± 2 mL/kg to 6 ± 1 mL/kg (mean ± SEM) at constant positive end-expiratory pressure. After restoration of initial V_T, we infused dobutamine to increase cardiac output ($\dot{Q}$) by the same amount as with hypercapnia. Permissive hypercapnia increased $\dot{Q}$ by an average of $1.4 \text{ L} \cdot \text{min}^{-1} \cdot \text{m}^2$, decreased arterial oxygen tension from 109 ± 10 mm Hg to 92 ± 11 mm Hg ($p < 0.05$), markedly increased true shunt ($\dot{Q}_S/\dot{Q}_T$), from 32 ± 6% to 48 ± 5% ($p < 0.0001$), and had no effect on the dispersion of $A_A/\dot{Q}/\dot{V}_A/\dot{Q}$. On reinstatement of baseline V_T with maintenance of a high $\dot{Q}$, $\dot{Q}_S/\dot{Q}_T$ remained increased, to 38 ± 6% ($p < 0.05$), and P_{aO_2}) remained decreased, to 93 ± 4 mm Hg ($p < 0.\ 05$). These results agreed with effects of changes in V_T and $\dot{Q}$ predicted by the mathematical lung model of the MIGET. We conclude that permissive hypercapnia increases pulmonary shunt, and that deterioration in gas exchange is explained by the combined effects of increased $\dot{Q}$ and decreased alveolar ventilation.

Ventilator-Induced Overdistension in Children: Dynamic versus Low-Flow Inflation Volume-Pressure Curves—Neve V, de la Roque ED, Leclerc F, Leteurtre S, Dorkenoo A, Sadik A, et al. Am J Respir Crit Care Med 2000 Jul;162(1):139-147.

We applied to 20 paralyzed ventilated children (0.15 to 14.3 yr, six with acute respiratory distress syndrome [ARDS]) the low-flow inflation (LFI) technique providing quasi-static volume-pressure (V-P) curves and compared the assessment of overdistension (OD) on dynamic and LFI (reference) inspiratory V-P curves. Dynamic curves were obtained at the airway opening during regular constant flow ventilation (Servo 300). Then LFI curves were obtained. Two analyses were performed: First, the nonlinear coefficient c of a second order polynomial equation (SOPE) fitted to dynamic data obtained during constant flow was compared with the c of SOPE fitted to LFI curve (within tidal volume [V_T]). Second, the dynamic C20/C (ratio of compliance of the last 20% of the curve (C20) to total compliance [C]) was compared with the determination of the upper inflection point (UIP) on the LFI curve. OD was defined as a negative value of c, a C20/C < 0.80, an UIP included within the V_T range for that child during regular ventilation. Using LFI V-P curves as reference, SOPE offered a better detection of OD than dynamic C20/C or the determination of the UIP by graphical means. Indeed the first analysis showed a substantial agreement ($\kappa = 0.75$) between dynamic c and LFI c detection of OD whereas the second analysis showed a poor agreement ($\kappa = 0.22$) between C20/ C and LFI detection of the UIP. In conclusion, quasi-static V-P curves can easily be obtained in children with the LFI technique. SOPE offers a good detection of OD on dynamic and LFI V-P curves but the C20/C index seems to be an inadequate measure of OD.

Airway Complications Following Pediatric Lung and Heart-Lung Transplantation—Kaditis AG, Gondor M, Nixon PA, Webber S, Keenan RJ, Kaye R, Kurland G. Am J Respir Crit Care Med 2000 Jul;162(1): 301-309.

Obstruction at the airway anastomosis is a recognized complication of adult-heart-lung transplantation (HLT) and lung transplantation (LT). Data for pediatric transplantation have been scarce. We reviewed our experience in pediatric HLT and LT to determine the frequency of airway complications and to document the therapeutic modalities used for their treatment. Fifty-three patients (median age: 13.8 yr; range: 1.3 to 28.2 yr) underwent HLT ($n = 25$), SLT ($n = 3$), DLT ($n = 25$), or repeat DLT ($n = 3$) and survived for more than 72 h. Major anastomotic airway complications requiring intervention affected one of the 25 HLT (4%) and seven of the 28 LT (SLT + DLT) patients (25%) ($p = 0.05$). Four patients with granulation tissue occluding the airway were treated with forceps resection, laser ablation, or balloon dilatation. Three patients with fibrotic strictures received silicone stents, laser ablation, or balloon dilatation. Two patients with bronchomalacia or diffuse stricture below the anastomosis underwent metal stent placement. Five of seven patients who were treated for anastomotic complications had satisfactory relief of airway obstruction. As compared with previously studied adults, pediatric heart-lung transplant recipients had the same or a lower frequency, and pediatric lung transplant recipients had a higher frequency of major anastomotic airway complications. A variety of treatment modalities were necessary to achieve adequate relief of airway obstruction.

A Randomized, Controlled Crossover Trial of Two Oral Appliances for Sleep Apnea Treatment—Bloch KE, Iseli A, Zhang JN, Xie X, Kaplan V, Stoeckli PW, Russi EW. Am J Respir Crit Care Med 2000 Jul;162(1):246-251.

Our purpose was to compare the effectiveness and side effects of a novel, single-piece mandibular advancement device (OSA-Monobloc) for sleep apnea therapy with those of a two-piece appliance with lateral Herbst attachments (OSA-Herbst) as used in previous studies. An OSA-Monobloc and an OSA-Herbst with equal protrusion were fitted in 24 obstructive sleep apnea patients unable to use continuous positive airway pressure (CPAP) therapy. After an adaptation period of 156 ± 14 d (mean ± SE), patients used the OSA-Monobloc, the-OSA-Herbst, and no appliance in random order, using each appliance for 1 wk. Symptom scores were recorded and sleep studies were done at the end of each week. Several symptom scores were significantly improved with both appliances, but to a greater degree with the OSA-Monobloc. Epworth Sleepiness Scale scores were 8.8 ± 0.7 with the OSA-Herbst, and 8.6 ± 0.8 with the OSA-Monobloc devices, and 13.1 ± 0.9 without therapy ($p < 0.05$ versus both appliances). The apnea/hypopnea index was 8.7 ± 1.5/h with the OSA-Herbst and 7.9 ± 1.6/h with the OSA-Monobloc device, and 22.6 ± 3.1/h without therapy ($p < 0.05$ versus both appliances). Side effects were mild and of equal prevalence with both appliances. Fifteen patients preferred the OSA-Monobloc, eight patients had no preference, and one patient preferred the OSA-Herbst device ($p < 0.008$ versus OSA-Monobloc). We conclude that both the OSA-Herbst and the OSA-Monobloc are effective therapeutic devices for sleep apnea. The OSA-Monobloc relieved symptoms to a greater extent than the OSA-Herbst, and was preferred by the majority of patients on the basis of its simple application.

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Biologically Variable or Naturally Noisy Mechanical Ventilation Recruits Atelectatic Lung—Mutch WAC, Harms S, Graham MR, Kowalski SE, Girling LG, Lefevre GR. Am J Respir Crit Care Med 2000 Jul;162(1):319-323.

Biologically variable mechanical ventilation ($\dot{V}$bv)—using a computer-controller to mimic the normal variability in spontaneous breathing improves gas exchange in a model of severe lung injury (Lefevre, G. R., S. E. Kowalski, L. G. Girling,-D. B. Thiessen, W. A. C. Mutch. Am. J. Respir. Crit. Care Med. 1996;154:1567-1572). Improved oxygenation with $\dot{V}$bv, in the face of alveolar collapse, is thought to be due to net volume recruitment secondary to the variability or increased noise in the peak inspiratory airway pressures (Ppaw). Biologically variable noise can be modeled as an inverse power law frequency distribution (y approximately $1/f^a$) (West, B. J., M. Shlesinger. Am. Sci.1990;78:40-45). In a porcine model of atelectasis—right lung collapse with one-lung ventilation—we studied if $\dot{V}$bv ($n = 7$) better reinflates the collapsed lung compared with conventional monotonously regular control mode ventilation ($\dot{V}$c; $n = 7$) over a 5-h period. We also investigated the influence of sigh breaths with $\dot{V}$c ($\dot{V}$s; $n = 8$) with this model. Reinflation of the collapsed lung was significantly enhanced with $\dot{V}$bv—greater P_{aO_2} (502 ± 40 mm Hg with $\dot{V}$bv versus 381 ± 40 mm Hg with $\dot{V}$c at 5 h; and 309 ± 79 mm Hg with $\dot{V}$s; mean ± SD), lower P_{aCO_2} (35 ± 4 mm Hg versus 48 ± 8 mm Hg and 50 ± 8 mm Hg), lower shunt fraction (9.7 ± 2.7% versus 14.6 ± 2.0% and 22.9 ± 6.0%), and higher respiratory system compliance (Crs) (1.15 ± 0.15 mL/cm H_2O/kg versus 0.79 ± 0.19 mL/cm H_2O /kg and 0.77 ± 0.13 mL/cm H_2O /kg)—at lower mean Ppaw (15.7 ± 1.4 cm H_2O versus 18.8 ± 2.3 cm H_2O and 18.9 ± 2.8 cm H_2O). $\dot{V}$bv resulted in an 11% increase in measured tidal volume (V_{Tm}) over that seen with $\dot{V}$c by 5 h (14.7 ± 1.2 mL/kg versus 13. 2 mL/kg). The respiratory rate variability programmed for $\dot{V}$bv demonstrated an inverse power law frequency distribution (y approximately $1/f^a$) with $a = 1.6 \pm 0.3$. These findings provide strong support for the theoretical model of noisy end-inspiratory pressure better recruiting atelectatic lung. Our results suggest that using natural biologically variable noise has enhanced the performance of a mechanical ventilator in control mode.

Availability of an Alcohol Solution Can Improve Hand Disinfection Compliance in an Intensive Care Unit—Maury E, Alzieu M, Baudel JL, Haram N, Barbut F, Guidet B, Offenstadt G. Am J Respir Crit Care Med 2000 Jul;162(1):324-327.

We investigated whether rubbing with an alcohol solution increases compliance with hand disinfection in a medical intensive care unit (MICU). During a first period (P1), hand disinfection was achieved only through conventional washing, whereas during a second period (P2), hand disinfection could be achieved either through conventional washing or rubbing with an alcohol solution. There were 621 opportunities for hand disinfection during P1 and 905 opportunities during P2. General compliance during P1 was 42.4%, and reached 60.9% during P2 ($p < 0.001$). This improvement was observed among nurses (45.3% versus 66.9%, $p < 0.001$), senior physicians (37. 2% versus 55.5%, $p < 0.001$), and residents (46.9% versus 59.1%, $p = 0.03$). Acceptability and tolerance were evaluated through the answers to an anonymous questionnaire distributed to all 53 health care workers in the MICU. Rubbing with alcohol solution was an easy procedure (100% of responses) and induced mild side effects in less than 10% of respondents. In a complementary study conducted 3 mo after the first one, compliance remained better than during P1 (51. 3% versus 42.4%, $p = 0.007$). These findings suggest that rubbing with alcohol solution increases compliance with hand disinfection, and that it could be proposed as an alternative to conventional handwashing in the MICU.

Nasal Continuous Positive Airway Pressure Reduces Sleep-Induced Blood Pressure Increments in Preeclampsia—Edwards N, Blyton DM, Kirjavainen T, Kesby GJ, Sullivan CE. Am J Respir Crit Care Med 2000 Jul;162(1):252-257.

Preeclampsia is the predominant cause of admissions to neonatal intensive care. The diurnal blood pressure pattern is flattened or reversed in preeclampsia. We hypothesized that snoring and partial upper airway obstruction contribute to nocturnal rises in blood pressure. We tested this hypothesis by controlling sleep-induced upper airway flow limitation and snoring with nasal positive pressure. Eleven women with preeclampsia underwent two consecutive polygraphic sleep studies with simultaneous beat-to-beat blood pressure monitoring. Average blood pressure for the night overall and in each sleep stage was calculated. Sleep architecture was similar on the two study nights. Sleep-induced partial upper airway flow limitation occurred in all patients in the initial study. Autosetting nasal continuous positive airway pressure (CPAP) applied at a mean maximal pressure of 6 ± 1 cm H_2O eliminated flow limitation throughout sleep on the treatment night. Blood pressure was markedly reduced on the treatment night [(128 ± 3)/(73 ± 3)] when compared with the initial nontreatment study night [(146 ± 6)/(92 ± 4)], p = (0.007)/(0.002). We conclude that partial upper airway obstruction during sleep in women with preeclampsia is associated with increments in blood pressure, which can be eliminated with the use of nasal CPAP.

Tracheal Gas Insufflation: Current Status and Future Prospects

A Special Issue

containing the papers from a conference held
August 19, 2000, in Dallas, Texas

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*Conference Co-Chairs

Tracheal Gas Insufflation

On August 19, 2000, the American Association for Respiratory Care (AARC) convened a state-of-the-art conference in Dallas, Texas, to review the technique tracheal gas insufflation (TGI). The topic is particularly relevant at this time for several reasons. First, with the recent publication of the Acute Respiratory Distress Syndrome Network Ventilator Management Trial,[1] showing that reduced lung distention during mechanical ventilation improved mortality, a dramatic increase in interest in "lung-protective ventilatory strategies" has developed. The potential ability of TGI to reduce dead space (and thus tidal volume) has thus become a conceptually attractive adjunct to ventilator management. Second, several investigators with industry backing are actively developing TGI systems. However, optimal design features, required monitoring capabilities, regulatory requirements, and various cost issues for these devices remain ill defined. Third, because so few clinical trials have been done using TGI, clinician awareness of the TGI concept seems lacking.

To address these issues, the American Association for Respiratory Care convened a unique collection of scientists, clinicians, industry representatives, and regulatory experts for this one-day conference. Speakers were charged to address specific issues on TGI, including design characteristics, safety/monitoring features, regulatory requirements, potential study designs, clinical applicability, and cost issues. Industry sponsors sent invited responders to ask questions and interact in discussions. The conference concluded with an interactive session addressing the barriers to the clinical implementation of TGI.

The proceedings of this conference comprise this special issue of Respiratory Care. We hope that readers of this issue will better understand TGI's potential role, as well as its potential problems, and also what needs to be done for it to enter clinical practice.

Neil R MacIntyre MD FAARC
Dean R Hess PhD RRT FAARC
Conference Co-Chairs

REFERENCE

1. Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. The Acute Respiratory Distress Syndrome Network. N Engl J Med 2000;342(18):1301–1308.

Tracheal Gas Insufflation and Related Techniques to Introduce Gas Flow into the Trachea

Dean R Hess PhD RRT FAARC and Michael A Gillette MD PhD

Introduction
Continuous Insufflation of Oxygen
Transtracheal Oxygen Administration
Transtracheal Jet Ventilation
High Frequency Jet Ventilation
Intratracheal Pulmonary Ventilation
Tracheal Gas Insufflation
Summary

Over the past 50 years, a variety of techniques have been developed that have in common the insufflation of gas into the central airway to facilitate carbon dioxide (CO_2) clearance. These include continuous insufflation of oxygen, transtracheal jet ventilation, high frequency jet ventilation, transtracheal oxygen administration, intratracheal pulmonary ventilation, and tracheal gas insufflation (TGI). Continuous insufflation of oxygen is a technique used to enhance CO_2 removal in the presence of apnea. Transtracheal jet ventilation and high frequency jet ventilation promote bulk gas flow into the lungs. Some techniques, such as transtracheal oxygen administration, provide insufflation of oxygen as an adjunct to spontaneous ventilation. However, other techniques, such as TGI, are used as an adjunct to positive pressure ventilation. Intratracheal pulmonary ventilation provides positive pressure ventilation while bypassing the upper airway. Although some of these techniques are promising adjuncts to mechanical ventilation and may help reduce ventilator-associated lung injury, much remains to be learned about their role in the care of patients with acute lung injury.

Key words: tracheal gas insufflation, transtracheal oxygen, transtracheal jet ventilation, high-frequency jet ventilation, intratracheal pulmonary ventilation, mechanical ventilation. [Respir Care 2001;46(2):119–129]

Introduction

Over the past 50 years, various techniques have been described that use a catheter to introduce a flow of gas into the trachea. These have in common a desire to enhance lung function by augmenting the clearance of carbon dioxide. A recent renascence of interest in these approaches has been spurred by the conjoint observations that overdistention of the lungs during positive pressure ventilation (PPV) can induce lung injury[1] and that ventilatory strategies that promote lung protection result in improved morbidity and mortality.[2,3] Since lung-protective strategies compromise ventilation, they may be facilitated by tech-

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A version of this report was given by Dr Hess at the special conference, Tracheal Gas Insufflation: Current Status and Future Prospects, presented by the American Respiratory Care Foundation, August 19, 2000, in Dallas, Texas.

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niques that enhance carbon dioxide (CO_2) clearance and thereby reduce the ventilatory requirement. In this paper, we describe various techniques that have been used to introduce gas flow into the trachea.

Continuous Insufflation of Oxygen

Apneic oxygenation is a technique in which the subject is hyperoxygenated by ventilation with 100% oxygen, and then has ventilation interrupted.[4,5] During the ensuing period of apnea, arterial partial pressure of oxygen (P_{aO_2}) is maintained above normal because of the very high P_{aO_2} at the initiation of apnea. The duration of apneic oxygenation is limited not by the decline in P_{aO_2}, but rather by the rise in arterial partial pressure of carbon dioxide (P_{aCO_2}). In 1961, Eger and Severinghaus[6] reported the rate of rise in alveolar partial pressure of carbon dioxide (P_{ACO_2}) in 5 healthy apneic anesthetized adult patients. In the first minute of apnea there was a rapid rise in P_{ACO_2} as the alveolar P_{CO_2} equilibrated with the mixed venous P_{CO_2}. This was followed by a linear rise in P_{CO_2} determined by the rate of tissue CO_2 production. The slope of the increase in P_{ACO_2} was 4 mm Hg/min without prior hyperventilation. The increase in P_{ACO_2} was less with prior hyperventilation, occurring at a rate of 3 mm Hg/min. At this rate of P_{CO_2} increase, the duration of apneic oxygenation was limited by the concomitant respiratory acidosis. From these and other studies published from the mid-1950s to the early 1970s, it was established that the rate of rise in P_{CO_2} during apnea in humans is about 3–5 mm Hg/min.[4]

It has been known for 50 years that continuous insufflation of oxygen (CIO) into the trachea or bronchi of experimental animals can slow the rate of P_{aCO_2} increase during apneic oxygenation. In 1951, Jacoby et al[7] reported that tracheal insufflation at 15 L/min slowed the rise in P_{CO_2} in apneic dogs. In the 1980s there were a number of published studies evaluating this technique in animal models.[8–17] With two catheters, one positioned 3.5 cm into each bronchus (Fig. 1), and a total flow of 40 L/min (20 L/min to each catheter), dogs can be maintained eucapnic during apnea.[18] This outcome has been attributed to the combined effects of convective streaming in the central airways due to the catheter flow and cardiogenically induced flow in the peripheral airways with molecular diffusion. Slutsky et al[19] studied insufflation of low flow ($\leq$ 3 L/min) oxygen 1 cm proximal to the carina in apneic dogs. They reported that the rate of increase in P_{aCO_2} decreased over time and reached a plateau at 2 hours (Fig. 2). The studies were stopped at 4–5 hours, with no dogs showing signs of cardiovascular or other compromise. However, P_{aCO_2} reached a level in excess of 160 mm Hg, associated with pH < 6.9 and $P_{aO_2} > 350$ mm Hg.

The first report of CIO in humans was by Comroe and Dripps in 1946.[20] They used tracheal flows of 6–11 L/min in two comatose patients and reported that P_{aCO_2} reached $>$ 300 mm Hg before arterial oxygen desaturation occurred! Babinski et al[21] used CIO in 5 adult apneic patients prior to operative procedures. Endobronchial catheters (Fig. 3) were placed and a total oxygen flow of 0.6–0.7 L/Kg/min was divided between the two catheters. Over 30 minutes, P_{aCO_2} rose from approximately 35 mm Hg to 55 mm Hg in 4 of the patients, remaining normal in one patient. Breen et al[22] delivered flows of 0.9–1.6 L/Kg/min to the bronchi of apneic anesthetized patients, and reported an increase in P_{aCO_2} over 30 minutes, from about 35 mm Hg to about 70 mm Hg. Perl et al[11] insufflated 0.5 L/Kg/min to the distal tip of a Carlen's tube in 5 apneic patients. They reported a 30% reduction in rate of increase of P_{aCO_2}, compared to apneic oxygenation alone (Fig. 4).

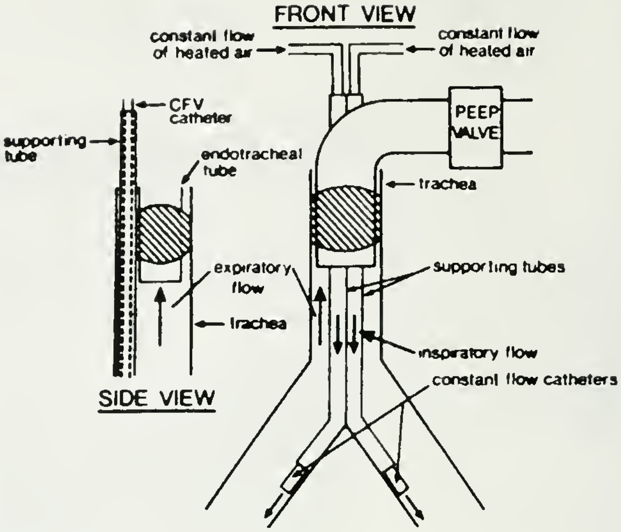

Fig. 1. Catheter position for continuous flow ventilation (CFV) in dogs. Note that the catheters are exterior to the endotracheal tube and that they extend into the mainstem bronchi. PEEP = positive end-expiratory pressure. (From Reference 18, with permission.)

Hypoxemia is a recognized complication of endotracheal suctioning. Most commonly, this complication is avoided by pre-oxygenation with 100% oxygen and limitation of procedure duration. CIO during suctioning has been suggested as an alternative method to prevent suctioning-related hypoxemia.[23–26] Smith et al[26] evaluated a double-lumen suction catheter, with oxygen insufflation through one lumen alternating with suction through the other. With this technique, they[26] were able to prevent hypoxemia during suctioning without pre-oxygenation. Brochard et al[25] evaluated CIO during suctioning using a specially designed endotracheal tube incorporating five 0.7 mm diameter capillaries into its wall. Gas from these capillaries exits 1 cm from the distal tip of the tube. Oxygen insufflation at 12 L/min minimized arterial desaturation during suctioning in severely hypoxemic patients with acute respiratory failure (ARF). Further, the decline in lung vol-

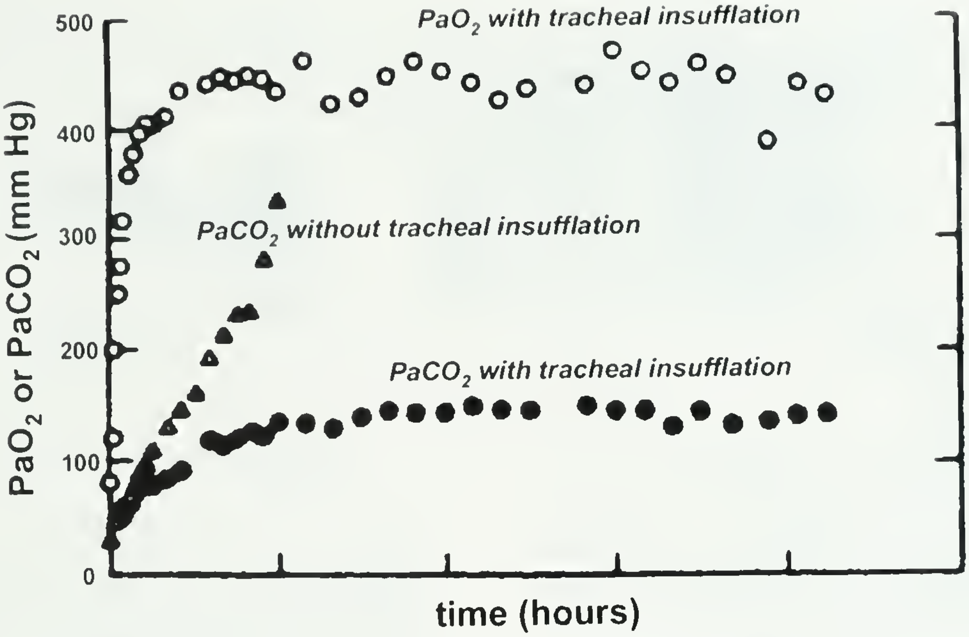

Fig. 2. Effect of tracheal insufflation of oxygen at 2 L/min in dogs. Note that the arterial partial pressure of oxygen (P_{aO_2}) remains high, that the arterial partial pressure of carbon dioxide (P_{aCO_2}) reaches a plateau with oxygen insufflation, and that the P_{aCO_2} rises with a rapid slope without oxygen insufflation. (From Reference 19, with permission.)

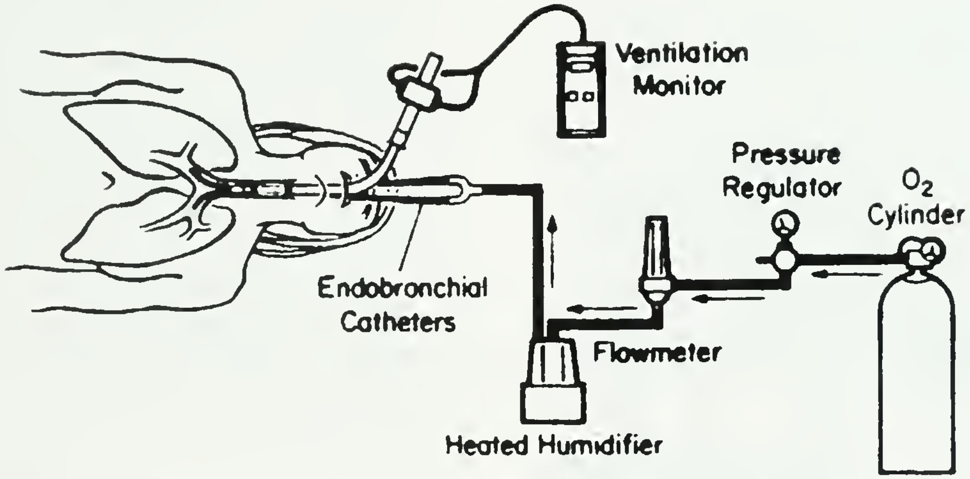

Fig. 3. Gas delivery system for continuous insufflation of oxygen in human subjects. (From Reference 4, with permission.)

ume that typically occurs with suctioning was ameliorated by use of oxygen insufflation. Although potentially beneficial, oxygen insufflation during suctioning has not become popular, probably because the closed suction system is commonly employed to minimize suction-related complications.

The necessity of ventilation during cardiopulmonary resuscitation (CPR) has been questioned.[27–30] Hallstrom et al[28] recently reported that the outcome after CPR with chest compressions alone was similar to that after chest compressions with mouth-to-mouth ventilation. Several studies have evaluated the use of CIO during CPR. In a canine model of cardiac arrest, Branditz et al[31] showed that hypercapnia did not occur and that arterial oxygen saturation was maintained using 15 L/min of transtracheal oxygen. Brochard et al[27] studied CIO at 15 L/min through an endotracheal tube in a porcine model of cardiac arrest. They noted similar P_{aO_2} and P_{aCO_2} with either standard CPR or oxygen insufflation CPR, the latter including chest compressions and insufflated oxygen without additional ventilation. Further, they found oxygen insufflation CPR led to improved hemodynamics relative to standard CPR. Saïssy et al[32] recently evaluated CIO during out-of-hospital CPR. Cardiac-arrested patients were randomized to stan-

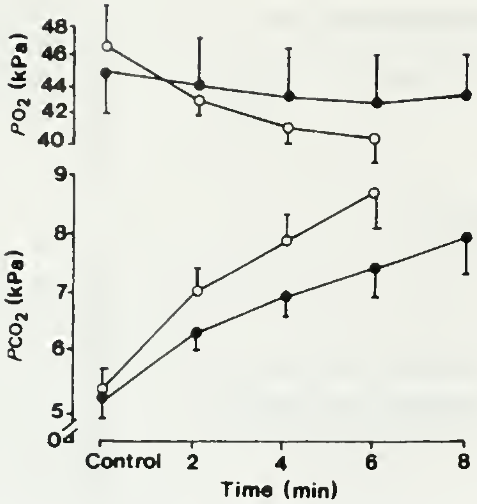

Fig. 4. Partial pressure of oxygen (P_{O_2}) and partial pressure of carbon dioxide (P_{CO_2}) with (closed circles) and without (open circles) continuous insufflation of oxygen in 5 patients during anesthesia. (From Reference 11, with permission.)

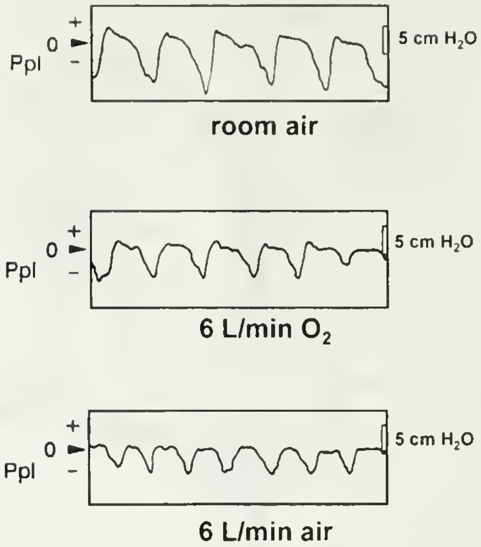

Fig. 6. Representative tracings of pleural pressure (Ppl) in a patient breathing room air with no transtracheal flow (top graph), while receiving transtracheal oxygen (middle graph), and while receiving transtracheal air (bottom graph). (From Reference 42, with permission.)

dard CPR or CIO plus chest compressions. They reported similar rates of successful resuscitation and levels of P_{aO_2} and P_{aCO_2} between the groups. CIO was provided through a specialized endotracheal tube with lateral channels in its wall allowing insufflation of oxygen at 15 L/min.

CIO has been studied as a technique to facilitate the determination of brain death in adults.[33–35] It is commonly recommended that 6 L/min of 100% oxygen be administered through a catheter placed at the level of the carina during this procedure to maintain adequate oxygenation.[34] With a starting P_{aCO_2} of 40 mm Hg and pre-oxygenation with 100% oxygen, the expected rise in P_{aCO_2} after initiation of apnea is 3–6 mm Hg/min (depending on tissue CO_2 production).

There are several reasons why CIO has not become widely accepted in the care of critically ill patients. Although this technique slows the rise in P_{aCO_2}, it does not provide adequate CO_2 clearance. This leads to the relatively rapid onset of unacceptable respiratory acidosis. Unlike the animals in which this technique has been studied, critically ill patients often are hypermetabolic and have elevated alveolar dead space, respectively increasing CO_2 production and decreasing CO_2 clearance. To maximize effective CO_2 clearance in CIO, high flows must be insufflated, producing the potential for airway injury. Critically ill patients also suffer elevated intrapulmonary shunt and require airway pressures greater than those provided by CIO alone in order to prevent alveolar derecruitment. Combined constant-flow and continuous PPV has been described, but only in an animal model.[14] The use of CIO is thus effectively limited to relatively short-term applications such as apnea testing to establish brain death, airway suctioning, or CPR.

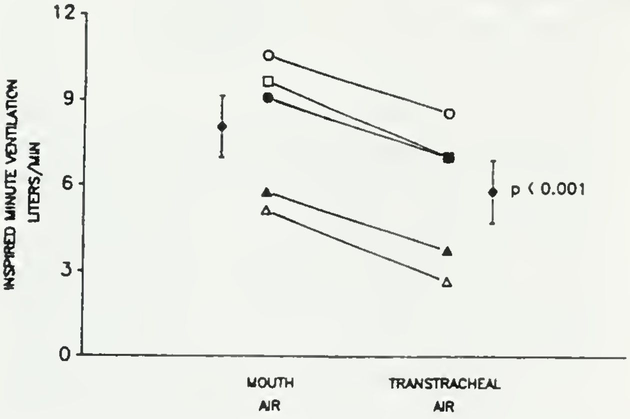

Fig. 5. Effect of transtracheal air on inspired minute ventilation of 7 patients. (From Reference 41, with permission.)

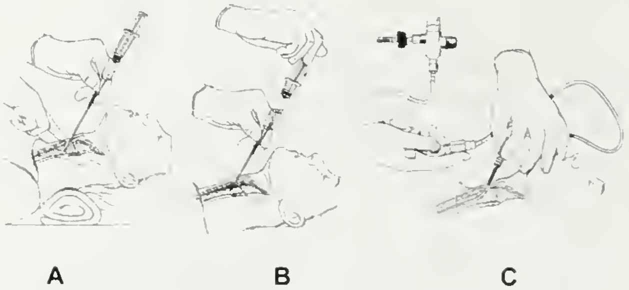

Fig. 7. Technique of percutaneous transtracheal jet ventilation. The needle is directed through the cricothyroid membrane (A), its position in the trachea is verified (B), and transtracheal ventilation is provided using a manual trigger (C). (From Reference 47, with permission.)

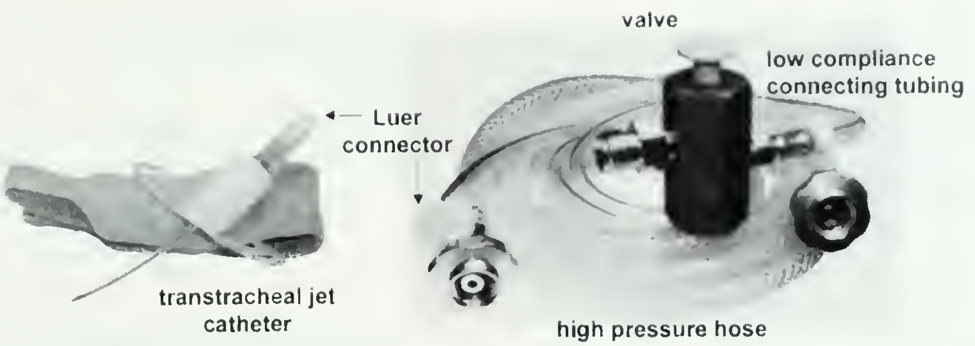

Fig. 8. Commercially available equipment for transtracheal jet ventilation: ACU 1060.1 transtracheal catheter (left) and BE 183-SUR manual jet ventilator (right). (Courtesy of Instrumentation Industries, Bethel Park, Pennsylvania.)

Transtracheal Oxygen Administration

Transtracheal oxygen administration delivers gas directly into the trachea via a small percutaneous catheter.[36] A principal advantage of transtracheal oxygen administration is its efficiency, allowing a reduction in flow and concomitant decrease in the cost of oxygen therapy. Even in the initial reports of transtracheal oxygen use, it was recognized that patients also frequently reported less dyspnea.[37,38] What was not initially recognized was that this was due to enhanced clearance of CO_2 from the upper airway—a tracheal gas insufflation (TGI) effect.

Bergofsky and Hurewitz[39] studied 5 patients who had chronic hypercapnia and permanent tracheostomies, in whom 5 L/min of gas was delivered through an otherwise occluded tracheostomy tube. They reported a significant reduction in dead space and inspired minute ventilation using this method. In one patient the effect was so dramatic that the patient requested long-term application of the technique. In a follow-up investigation, Hurewitz et al[40] studied patients with transtracheal oxygen catheters and reported that increases in oxygen flow from 1–8 L/min produced progressive decreases in dead space volume, tidal volume (V_T), and inspired minute ventilation.

Couser and Make[41] studied 7 patients receiving oxygen therapy via transtracheal catheter. As the transtracheal oxygen flow increased, inspired minute ventilation decreased; at 6 L/min inspired minute ventilation was reduced by approximately 50%. To demonstrate that this effect was due to gas flow and not to oxygen supplementation, transtracheal air was substituted for oxygen with a similar reduction in minute ventilation (Fig. 5). Benditt et al[42] studied 5 patients receiving transtracheal oxygen therapy and reported that the tension-time index of the diaphragm decreased as transtracheal flow of oxygen or air increased (Fig. 6). These data are consistent with a reduced ventilatory requirement with transtracheal gas flow, presumably due to a reduction in dead space.

Transtracheal oxygen insufflation has also been used for the treatment of obstructive sleep apnea.[43–45] In 5 patients with tracheostomies placed for the treatment of severe obstructive sleep apnea, Schneider et al[45] found that 15 L/min of tracheal oxygen insufflation stabilized the breathing pattern by providing sufficient air flow for the patients to inspire during upper airway obstruction. They also reported that this flow resulted in laryngeal obstruction during transitional sleep, which could result in high tracheal pressure unless a pressure pop-off is used.

Transtracheal Jet Ventilation

Percutaneous transtracheal jet ventilation (TTJV) is a technique in which a large intravenous catheter is inserted through the cricothyroid membrane and ventilation is pro-

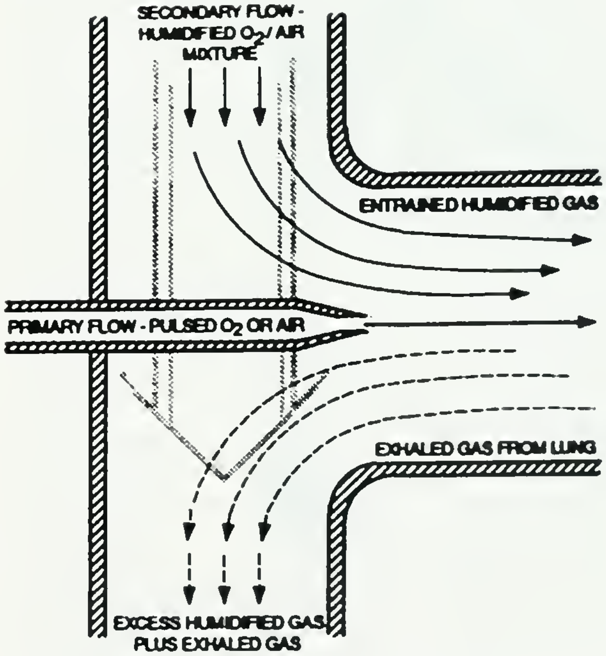
SECONDARY FLOW - HUMIDIFIED O2/AIR MIXTURE
ENTRAINED HUMIDIFIED GAS
PRIMARY FLOW - PULSED O2 OR AIR
EXHALED GAS FROM LUNG
EXCESS HUMIDIFIED GAS PLUS EXHALED GAS

Fig. 9. High frequency jet ventilation. The jet effect at the proximal airway entrains flow from a secondary gas source. The tidal volume delivered is the combination of the primary jet flow and the entrained gas flow. (From Reference 51, with permission.)

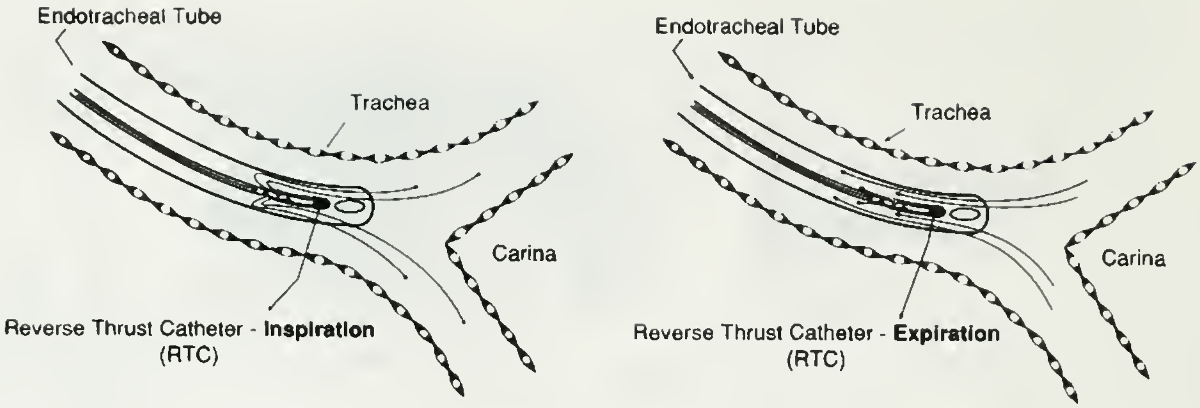
Endotracheal Tube
Trachea
Carina
Reverse Thrust Catheter - **Inspiration**
(RTC)
Endotracheal Tube
Trachea
Carina
Reverse Thrust Catheter - **Expiration**
(RTC)

Fig. 10. The reverse-thrust catheter used for intratracheal pulmonary ventilation (ITPV). During inspiration (left) the expiratory valve is closed and thus all of the gas flow is directed into the lungs. During expiration (right), the expiratory valve is open and the catheter flow and expiratory flow from the patient's lungs are expired simultaneously through the endotracheal tube. (From Reference 64, with permission.)

vided from a high-pressure gas source[46,47] (Fig. 7). In addition to a 50 psi gas source and a 14–16 gauge catheter, the TTJV system requires low-compliance connecting tubing and a valve to control flow time to the catheter.[46,47] Valve systems are commercially available (Fig. 8). The chief advantage of TTJV is that it is quicker and simpler than percutaneous cricothyroidotomy. A syringe containing 10 mL of saline is attached to the catheter, which is advanced through the cricothyroid membrane into the tracheal lumen. Free return of air confirms catheter tip position. The catheter is advanced over the introducer needle into the trachea and attached to the flow-controlling valve. The catheter is held in place manually to avoid displacement. Ventilation is provided at a rate of 12–20 cycles per minute.

Flow through a 16-gauge catheter at a driving pressure of 50 psi is about 500 mL/s. Because of the high velocity of gas exiting the catheter in the trachea, additional flow may be entrained from the upper airway. The peak inspiratory pressure during TTJV depends on the cross-sectional area of the trachea, the length and diameter of the catheter, the degree of upper airway obstruction, the compliance of the respiratory system, and the inspiratory time. Because exhalation occurs through the upper airway, complete up-

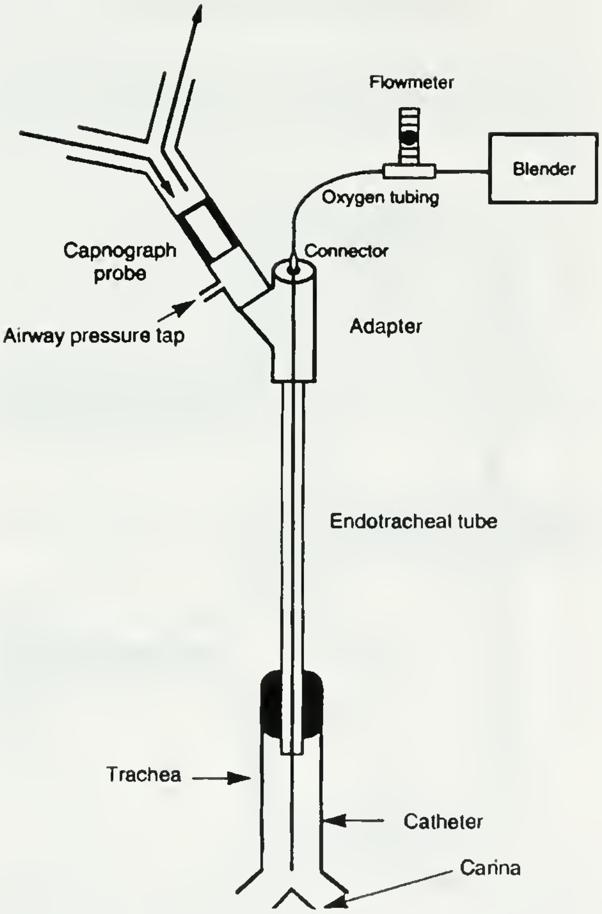

Fig. 11. A simple circuit for tracheal gas insufflation. (From Reference 71, with permission.)

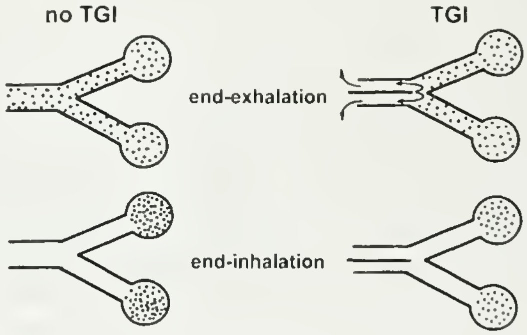

Fig. 12. With no tracheal gas insufflation (TGI) (left), the central airways contain CO_2 at end-expiration and this CO_2 is delivered to the alveoli during the subsequent inspiration. With TGI (right), the CO_2 from the central airways is flushed during the expiratory phase, which reduces the CO_2 delivered to alveoli during the subsequent inspiration. (From Reference 72, with permission.)

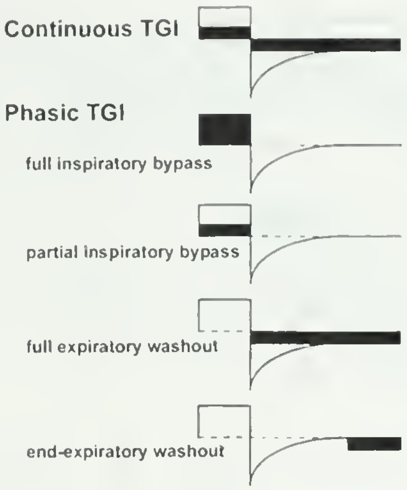

Fig. 13. Tracheal gas insufflation (TGI) can be provided as a continuous flow of gas into the airway or specific to the phase of the respiratory cycle. With phasic TGI, various approaches can be used. (From Reference 72, with permission.)

per airway obstruction is a contraindication to TTJV. Reported complications of TTJV are infrequent; they include hemorrhage at the insertion site, subcutaneous and mediastinal air, esophageal injury, and pneumothorax.[48]

Patel[48] reported 5 years' experience with TTJV in 29 patients. Two important benefits of TTJV were suggested. First, TTJV provided adequate oxygenation, allowing time for definitive airway management. Second, TTJV improved visualization of the glottic aperture, thereby promoting subsequent translaryngeal intubation.

High Frequency Jet Ventilation

High-frequency jet ventilation (HFJV) is a form of life support in which high velocity jets of gas are injected into the airway at frequencies higher than those used with conventional ventilators (100–660 per minute).[49,50] The driving pressure applied to the jet is typically 35–50 psi. This technique was originally applied using a specialized endotracheal tube incorporating a lumen designed to permit passage of the gas stream. Because of the undesirability of reintubating critically ill patients, techniques allowing the jet stream to be applied to the proximal endotracheal tube were subsequently introduced. The volume of gas delivered through the jet is relatively small (2–5 mL/Kg). However, because of the high velocity at which gas exits the jet, additional gas is entrained (Fig. 9), resulting in estimated V_T of 3–5 mL/Kg. The construction of these jet ventilation devices virtually precludes measurement of exact V_T. With HFJV, alveolar ventilation depends on the driving pressure to the jet, jet frequency, inspiratory-to-expiratory ratio, and overall pulmonary mechanics.

The efficacy of HFJV for patients with acute respiratory distress syndrome has been disappointing. Carlon et al[52] reported that HFJV provided no better oxygenation and ventilation than conventional techniques. Further, survival was not improved with the use of HFJV. Gluck et al[51] reported improved gas exchange and reduced airway pressure during a 24-hour trial of HFJV. As this study was uncontrolled, however, it is impossible to know whether similar effects could have been achieved with conventional ventilation. In addition, the short-term nature of the study prevented assessment of such important outcomes as survival.

Although HFJV has been virtually abandoned in the adult intensive care unit, it is used occasionally in neonates and in the operating room. In many neonatal intensive care units, high-frequency oscillatory ventilation is now used in place of HFJV. In the operating room, HFJV is used during head and neck surgery, allowing the patient to be oxygenated and ventilated without a large endotracheal tube obscuring the airway.[53]

Concerns related to HFJV include adequate humidification of the jet gas flow[54] and intrinsic positive end-expiratory pressure due to the short expiratory time.[55] Tracheal injury following HFJV in neonates has been reported.[56–58]

Intratracheal Pulmonary Ventilation

Intratracheal pulmonary ventilation (ITPV) is a technique that introduces a continuous-flow catheter into the endotracheal tube.[59–63] The distal tip of the catheter is positioned about 1 cm from the carinal tip of the endotracheal tube. A unique, reverse-thrust catheter design allows gas exiting the distal end of the catheter to be directed cephalad (Fig. 10). The exhalation port of the endotracheal tube is attached to the ventilator. When the exhalation valve is closed, flow is delivered into the distal lungs. When the exhalation port is open, the reverse-thrust catheter entrains gas from the distal airways to facilitate exhalation.

The objective of ITPV is to reduce V_T and thus alveolar distending pressure. This may be achieved by several mechanisms associated with this technique. First, the V_T is delivered directly into the trachea, bypassing the proximal dead space. Second, the proximal dead space is flushed during the expiratory phase. Third, the jet effect of the reverse-thrust catheter facilitates gas flow from the distal lungs. An additional effect of ITPV that has been reported is augmented clearance of mucus from the inner lumen of the endotracheal tube.[65] Most ITPV research has occurred in animal models.[60–62,64,66] The reported human experience has been limited primarily to neonatal and pediatric patients,[67–69] although its use for patients with acute respiratory distress syndrome has been recently reported.[63]

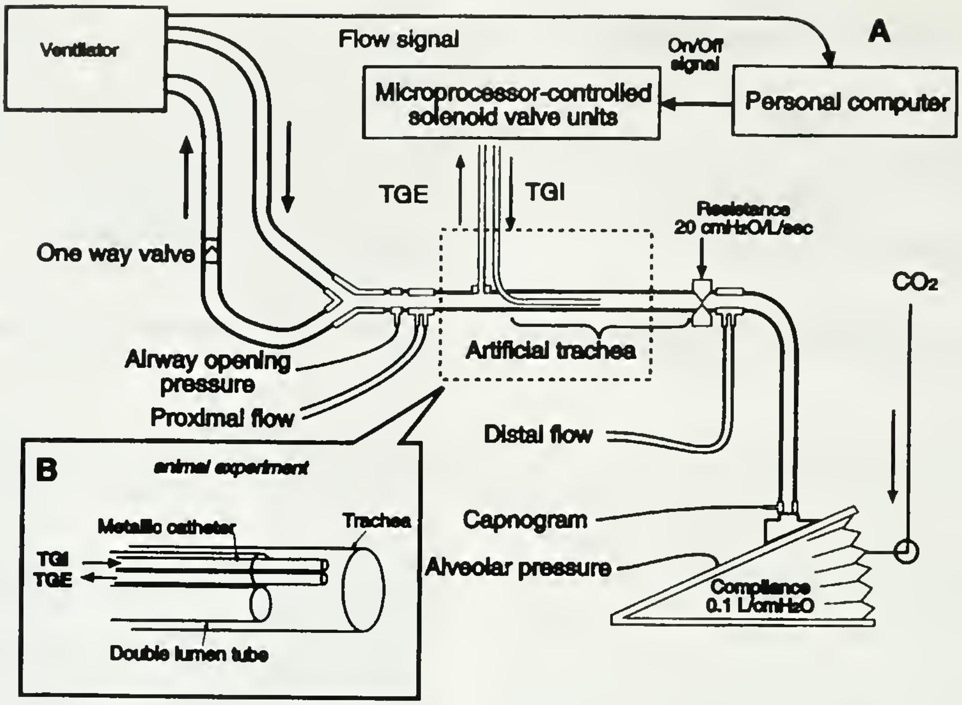

Fig. 14. Schematic of a system for tracheal gas insufflation (TGI) with tracheal gas exsufflation (TGE). (From Reference 99, with permission.)

The future role of ITPV in adult patients with ARF remains to be determined.[59–71]

Tracheal Gas Insufflation

TGI is the injection of fresh gas into the central airways for the purpose of improving the efficiency of alveolar ventilation and/or minimizing the ventilatory requirement.[71,72] It is used as an adjunct to mechanical ventilation. A catheter is placed into the central airway proximal to the carina (Fig. 11). Flow is introduced through the catheter to flush the proximal airways of CO_2-laden gas (Fig. 12). The result is less CO_2 rebreathing on the subsequent inspiration, which effectively lowers the dead space. TGI has been studied extensively in lung models[73–75] and animals.[76–88] In recent years there have been an increasing number of reports of the use of TGI for patients with ARF.[89–97] As it has become more widely recognized that alveolar overdistention during mechanical ventilation may result in increased morbidity and mortality,[3] there is considerable academic and clinical interest in TGI as a technique to reduce the ventilatory requirement of patients with ARF.

A variety of approaches to TGI have been reported. The catheter can be introduced into the trachea either beside the endotracheal tube or through the endotracheal tube, or can be incorporated into the endotracheal tube design. Catheters can introduce flow either toward the carina or away from the carina (retrograde or reverse-thrust catheters). The flow can be continuous throughout the respiratory cycle, restricted to the expiratory phase (Fig. 13), or constrained to a specific portion of the expiratory phase. Regardless of the approach that is used, a concern with the use of TGI is the interaction between the TGI flow and the ventilator.[71]

A recently described alternative to TGI is aspiration of airway dead space—tracheal gas exsufflation (TGE).[98] With this technique, airway dead space gas is aspirated from the distal endotracheal tube and replaced by fresh gas from the ventilator circuit. Potential advantages of this approach are elimination of TGI-related problems such as airway injury due to jet streams from the catheter and difficulties with humidification of the TGI gas flow. Takahashi et al[99] described the effects of combined TGE and TGI, in which TGE is applied early in the expiratory phase and TGI is applied late in the expiratory phase (Fig. 14). In a lung model and in experimental animals, they reported that combining TGE and TGI allowed precise control of end-expiratory lung volume and effective CO_2 elimination.

Summary

A variety of techniques have been developed that have in common the insufflation of gas into the central airway to facilitate CO_2 clearance. Some of these techniques are used to enhance CO_2 removal in the presence of apnea (eg, CIO). Others are used to promote bulk gas flow in the lungs (eg, TTJV and HFJV). Some are used as an adjunct to spontaneous ventilation (eg, transtracheal oxygen administration), whereas others are used as an adjunct to PPV (eg, TGI). Some provide PPV while bypassing the upper airway (eg, ITPV). Although some of these techniques are promising adjuncts to mechanical ventilation and may help reduce ventilator-associated lung injury (eg, TGI), much remains to be learned about their role in the care of patients with acute lung injury.

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Ventilator-Induced Lung Injury and the Evolution of Lung-Protective Strategies in Acute Respiratory Distress Syndrome

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Introduction
Pathophysiology of Acute Respiratory Distress Syndrome
Traditional Mechanical Ventilation in Acute Respiratory Distress Syndrome: Strategies and Complications
  Conventional Barotrauma
  Systemic Gas Embolization
  Ventilator-Induced Lung Injury
  Normal Lungs
  "Barotrauma" versus "Volutrauma"
  Injured Lungs
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  Low Tidal Volume Injury and Positive End-Expiratory Pressure
Contemporary Mechanical Ventilation in Acute Respiratory Distress Syndrome: Lung Protection and Permissive Hypercapnia
  Physiologic Consequences of Permissive Hypercapnia
  Clinical Consequences of Permissive Hypercapnia
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Beyond Acute Respiratory Distress Syndrome
Summary

Traditional ventilator management of acute respiratory distress syndrome (ARDS), emphasizing normalization of blood gases, promoted high rates of conventional barotrauma. Research revealed a broader range of ventilator-induced lung injury, physiologically and histopathologically indistinguishable from ARDS itself. It is now known that overdistention and cyclic inflation of injured lung can exacerbate lung injury and probably promote systemic inflammation, effects minimized by low tidal volumes/plateau pressures and by application of positive end-expiratory pressure. No compelling data suggest a safe interval for nonprotective ventilation in humans; historically defined "low" tidal volumes may remain excessive for certain patients. Protective ventilation, however, entails carbon dioxide accumulation ("permissive hypercapnia"). Despite extensive study, debate remains, even over whether consequent respiratory acidosis is harmful, tolerable with physiologic adaptation, or intrinsically adaptive. Its gross systemic effects seem generally tolerated by critically ill patients; however, subsets, including those with ischemic heart disease, left or right heart failure, pulmonary hypertension, or cranial injury, may be at higher risk. In controlled trials demonstrating mortality benefit from lung-protective ventilation, acidosis was more tightly controlled than in negative studies. Decreased acidosis-associated dyspnea probably explains reduced use of sedatives and paralytics noted in those trials. There may thus be disparate goals in ARDS management: rapid institution of a restrictive ventilatory strategy, and avoidance of significant acidosis. We review data pertaining to ARDS physiology, ventilator-induced lung injury, lung-protective ventilatory strategies, and the physiology of respiratory acidosis. Tracheal gas insufflation is considered as a means to reconcile the clinical goals of ventilatory reduction and control of acidosis.

Key words: tracheal gas insufflation, ventilator-induced lung injury, barotrauma, volutrauma, mechanical ventilation, acute respiratory distress syndrome, ARDS, respiratory acidosis, permissive hypercapnia. [Respir Care 2001;46(2):130–148]

Introduction

Tracheal gas insufflation (TGI) has been proposed as an adjunct to mechanical ventilation, especially in clinical settings demanding a low tidal volume (V_T) "lung-protective" approach, in which carbon dioxide accumulation is an issue. Concerns over ventilator-induced lung injury and lung-protective ventilatory strategies are particularly relevant to the management of acute respiratory distress syndrome (ARDS). This article reviews the evidence in support of evolving lung-protective strategies, with special reference to ARDS, and examines the clinical importance of permissive hypercapnia. Additional applications of TGI to clinical care are also considered.

Pathophysiology of Acute Respiratory Distress Syndrome

The appellation "respiratory distress syndrome" was first applied to adults by Ashbaugh et al in 1967.[1] Although it had been recognized for decades that profound physiologic disturbances such as trauma, sepsis, and pancreatitis could lead to severe pulmonary pathology, it was not until Ashbaugh's landmark study that the process was recognized as a discrete entity with clinical and pathologic features closely reminiscent of those in infantile respiratory distress syndrome. Manifestations in the original description included "severe dyspnea, tachypnea, cyanosis refractory to oxygen therapy, loss of lung compliance, and diffuse alveolar infiltration seen on chest x-ray." Initial ventilator support was pressure-cycled, but this approach "would not deliver adequate volumes at the high pressures required," and subsequent support was volume-cycled. On the respiratory support strategy adopted by Ashbaugh, only 4 of 12 patients had arterial partial pressure of carbon dioxide (P_{aCO_2}) > 45 mm Hg; the highest P_{aCO_2} measured was 63 mm Hg. Overall mortality was 58%. Notably, the single therapeutic intervention beyond general supportive care that appeared to be helpful was the application of positive end-expiratory pressure (PEEP).

Thirty-four years later, Ashbaugh's initial description remains remarkably apt. While the diagnostic requirements of ARDS have been only slightly refined,[2] the pathophysiologic derangements have become substantially better characterized over the intervening decades (Fig. 1). The proximal inciting event appears to be pulmonary vascular endothelial injury by inflammatory mediators, radical oxygen species, and other toxic substances elaborated by activated inflammatory cells. Positron emission tomography scanning suggests that this increased vascular permeability may initially be relatively uniform throughout the lung.[4] Alveolar capillary membrane disruption leads to accumulation of proteinaceous noncardiogenic edema fluid in the air spaces, a histopathologic picture referred to as diffuse alveolar damage. Secondary surfactant dysfunction further impairs gas exchange. Edematous lung promotes collapse of dependent lung units. Thus, while the plain radiographic picture, like the positron emission tomography scan, shows a diffuse bilateral process, higher resolution computed tomography scans reveal marked underlying heterogeneity, with areas of consolidation, particularly in dependent regions, interspersed with areas of normal-appearing lung.[5–7] If unchecked, the process can progress to a fibroproliferative phase, marked by irregular collagen deposition and permanent loss of compliance. Overall mortality from ARDS remains high, ranging from 40% to 50% in most studies.

Important insights have evolved from this detailed characterization of the pathophysiology of ARDS. Though overall pulmonary radiographic density is increased in ARDS patients, radiographically spared areas retain normal compliance.[8] This has led to the concept of ARDS-affected lungs as small rather than stiff, characterized by fewer functional alveoli rather than diffusely decreased compliance—the "baby lung" model.[7] Computed tomography scanning during tidal breathing suggests that a 3-compartment model may best depict the lung during early ARDS. In addition to an essentially normal compartment and an irremediably consolidated compartment, there is a compartment that is "recruitable," collapsed at low airway pressures but inflating with sufficient distending pressures. During tidal breathing at low levels of PEEP, this compartment can be observed to collapse and reinflate cyclically.[9] PEEP improves oxygenation to a degree proportional to the amount of this recruitable lung,[10] accounting in part for Ashbaugh's original observation of its benefits.[1] With increased airway pressure, recruitable lung expands, but nondependent, normal alveolar regions are prone to overdistention (Fig. 2). Static compliance increases in the recruited lung regions but decreases in the overdistended lung, leading to a redistribution of ventilation.[11] These basic characteristics of pulmonary physiology in the ARDS patient inform the following discussion of the evolution of appropriate ventilatory strategies.

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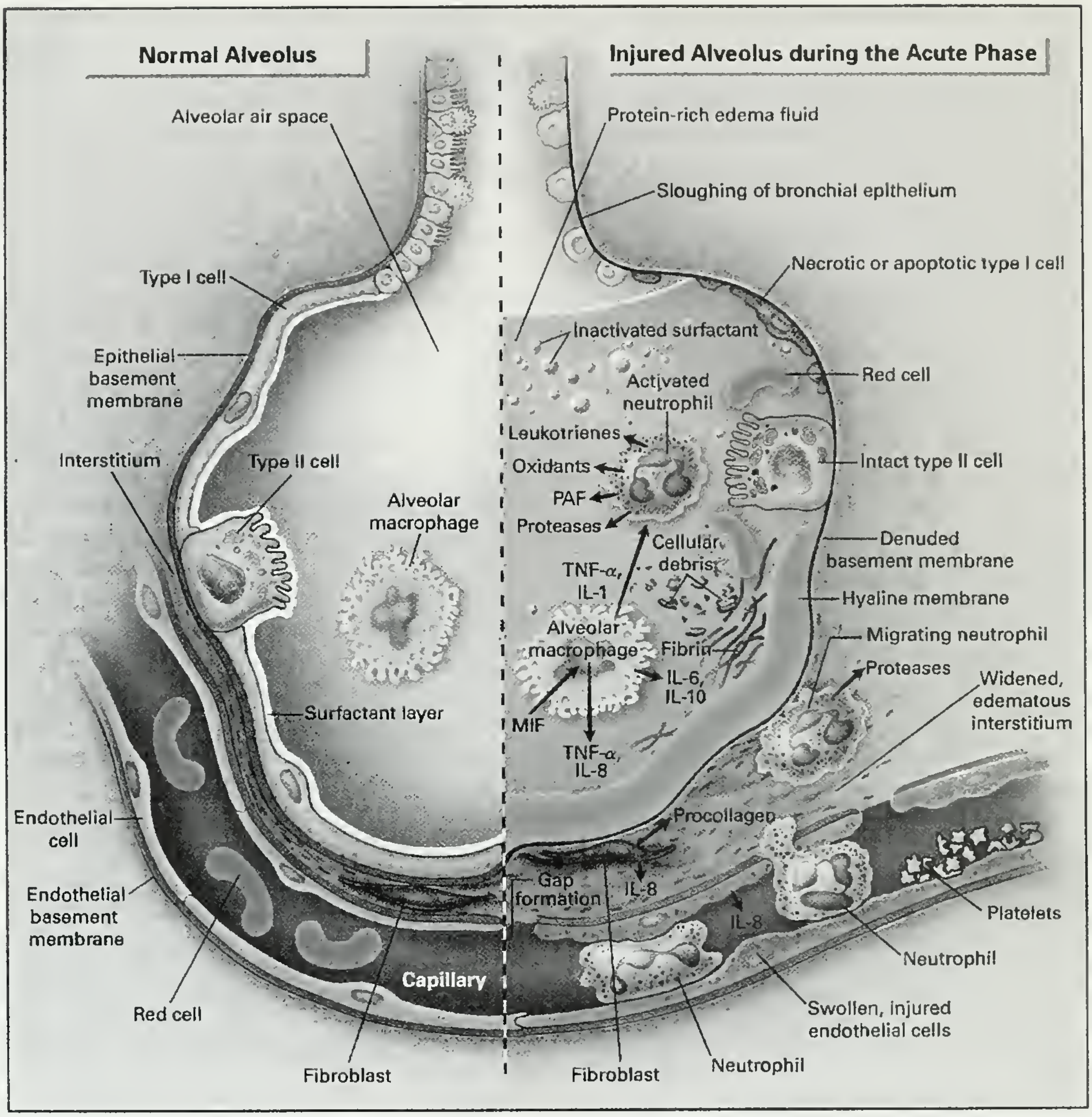

Fig. 1. Normal alveolus (left) and injured alveolus in the acute phase of the acute respiratory distress syndrome (right). The inciting event is thought to be injury of pulmonary vascular endothelium. Bronchial and alveolar epithelium sloughs and contributes to hyaline membrane formation. Neutrophils marginate through interstitium into air space, where they release oxidants, proteases, and other proinflammatory molecules. Alveolar macrophages secrete cytokines, interleukins, and tumor necrosis factor α, stimulating chemotaxis and activating neutrophils, promoting a cycle of inflammation and injury. Protein-rich edema fluid fills the alveolus, contributing to surfactant dysfunction. IL = interleukin. PAF = platelet-activating factor. MIF = macrophage inhibitory factor. (From Reference 3, with permission.)

Traditional Mechanical Ventilation in Acute Respiratory Distress Syndrome: Strategies and Complications

Although in Ashbaugh's original series, 4 of 12 ARDS patients were not mechanically ventilated at the time of diagnosis, all required mechanical ventilation during the courses of their diseases. Indeed, while not part of the formal definition, the need for mechanical ventilatory support is virtually a sine qua non for ARDS. Important advances have been made, and continue to be made, in determining the risks and benefits of such support and in improving the safety and efficacy of mechanical ventilation.

"Traditional" ventilator management in ARDS, as in other causes of respiratory failure, emphasized unloading of respiratory musculature and normalization of blood gases. The fraction of inspired oxygen (F_{IO_2}) was titrated to provide oxygen saturations in the range of 88–95%.

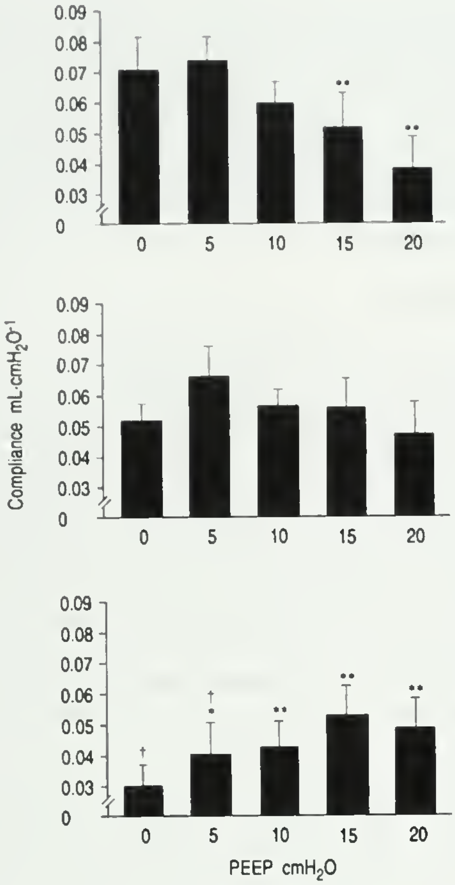

Fig. 2. Compliance distributed in ventral (upper graph), middle (middle graph), and dorsal (lower graph) lung in a supine acute respiratory distress syndrome patient, at 5 different levels of positive end-expiratory pressure (PEEP). Vertical comparisons: In relatively spared upper lung, compliance is essentially normal. Compliance trends toward moderate reduction in the middle region. Compliance is markedly reduced in the lower region († = $p < 0.01$). Horizontal comparisons: In relatively normal upper lung, increasing PEEP leads to overdistention and decreased compliance. In middle lung, there is no change in compliance with PEEP. Lower lung units are recruited with PEEP, increasing compliance (* = $p < 0.05$, ** = $p < 0.01$). Data are mean ± SEM. (From Reference 7, with permission.)

PEEP was used variably, but typically increased with increasing F_{IO_2} requirement.[12] Supraphysiologic V_T in the range of 10–15 mL/kg was standard,[13,14] based in large part on an anesthesia literature suggesting that microatelectasis and worsening gas exchange resulted from shallower breaths. Respiratory rate was then adjusted to provide minute ventilation adequate to normalize P_{aCO_2} and pH. Often the high pressure requirements of this strategy necessitated volume-cycled modes of ventilation.

Conventional Barotrauma

Traditional ventilatory strategies, though frequently capable of achieving their express goals of near normal arterial partial pressure of oxygen (P_{aO_2}) and P_{aCO_2}, were associated from the start with well-defined hazards. Initial concerns emphasized a range of clinically and radiographically apparent lung injury collectively referred to as "barotrauma." This familiar phenomenon, studied in detail by Macklin and Macklin,[15] is characterized by gas, escaped from ruptured alveoli, tracking through bronchovascular sheaths and adjoining tissue planes to yield pulmonary interstitial emphysema, subcutaneous emphysema, pneumomediastinum, and pneumothorax as its principal macroscopic manifestations. Experimental overinflation of fresh cadaver lungs suggested different profiles of pressure excess in the pathogenesis of different patterns of barotrauma. Intermittent excessive pressures resulted in the rupture of peripheral alveoli and pneumothorax, while less extreme but more sustained pressure elevations led to pneumomediastinum and subcutaneous emphysema.[16] Both pressure profiles could be seen in traditional ventilatory management.

Barotrauma occurred at widely varying rates in different series of mechanically ventilated patients, but patterns of predisposition began to emerge. In a prospective study of 354 patients undergoing assisted ventilation, Zwillich et al reported an overall pneumothorax incidence of 4.2%.[17] There were significant associations with younger age and use of PEEP. Ventilatory pressures were not noted. Barotrauma incidence was similar in a subsequent prospective study of 553 patients receiving ventilator support.[18] Although the overall rate was 4%, patients undergoing volume-cycled ventilation had a barotrauma frequency of 9%, significantly higher than the 1% rate associated with pressure-cycled ventilation. Use of PEEP increased barotrauma to 15%; the explicit indication for PEEP was an F_{IO_2} requirement > 0.5 to maintain $P_{aO_2} > 60$ mm Hg. Petersen and Baier found an 8% rate of pneumomediastinum, subcutaneous emphysema, or pneumothorax in an exclusively medical intensive care population. Those with barotrauma were younger, had higher peak inspiratory pressure (PIP), and had higher levels of maximum PEEP.[19] Indeed, pulmonary barotrauma incidence was 43% with PIP > 70 cm H_2O, 8% with PIP of 50–70 cm H_2O, and 0% with PIP < 50 cm H_2O. Although the authors did not specifically note the association, half of the patients with barotrauma carried a diagnosis of ARDS. This diagnostic association implicit in the aforementioned studies was made explicit by Gammon et al in a retrospective analysis of 139 intubated patients.[20] Mediastinal emphysema or pneumothorax was seen in 24% of patients overall, but in 66% of ARDS patients. Patients with barotrauma had higher PIP, PEEP, respiratory rate, and minute ventilation than patients without this complication; however, these associations were largely due to their cosegregation with the higher-risk ARDS population.

Systemic Gas Embolization

Gas escaping from injured lung need not be contained in extra-alveolar spaces of the thorax. A further complication, described in adults by Marini and Culver,[21] is systemic embolization of gas via a bronchovenous connection, leading in severe cases to a syndrome of livedo reticularis, cerebral infarction, and myocardial injury. Akin to its occurrence in diving accidents, microembolization of gas impedes blood flow through small vessels mechanically and by induced vasospasm, promoting tissue ischemia. While the small quantities and rapid resorption of embolized gas make definitive diagnosis singularly difficult, Marini and Culver speculated that in certain clinical contexts such unexplained conditions as sudden agitation, delirium, and local pain might be ascribable to this complication of mechanical ventilation.[21]

Ventilator-Induced Lung Injury

While subcutaneous emphysema, pneumothorax, pneumomediastinum, and perhaps systemic gas embolism are important causes of morbidity and mortality in mechanically ventilated patients, manifest air leaks do not comprehend the spectrum—indeed, are probably not the most important form—of what has come to be known as ventilator-induced lung injury (VILI). A great deal of attention has focused in recent years on data, particularly from animals, showing that mechanical ventilation can cause a pattern of diffuse lung injury indistinguishable from the histopathology characteristic of early ARDS.

The possibility that high-pressure ventilation of lungs with heterogeneous compliance characteristics could itself cause hemorrhage and hyaline membrane formation was suggested by Mead et al.[22] Teplitz extended this reasoning to suggest that ARDS might be considered an iatrogenic process due to the aggressive mechanical ventilatory support of patients with noncardiogenic pulmonary edema.[23] He observed that pulmonary pathology specimens collected from burn patients prior to 1963 rarely showed hyaline membrane formation, whereas hyaline membranes became more common after 1963. This distinction correlated with the development of routine clinical blood gas monitoring, and hence with the institution of more vigorous interventions in patients with severe gas exchange abnormalities. These speculations were provocative and insightful, and while few would argue that ARDS is fundamentally iatrogenic, there is now widespread agreement that mechanical ventilation can contribute to this subtler but profound form of lung injury.[24,25]

A plethora of animal studies have amassed demonstrating that lung injury can be exacerbated, or created de novo, under certain patterns of mechanical ventilation. Insights have been gained into the nature of alterations in pulmonary capillary permeability, the relative importance of extremes of pressure and volume, the role of cyclic recruitment and derecruitment, and the respective contributions of PEEP and V_T. Since these issues lie at the very heart of contemporary volume-limited ventilatory strategies, they bear further consideration here. VILI has been comprehensively reviewed in a recent report by Dreyfuss and Saumon.[25]

Normal Lungs

Greenfield et al were among the first to demonstrate that mechanical ventilation at high airway pressures was associated with lung damage. They exposed healthy dogs to PIP of 26–32 cm H_2O over 2 hours. Twenty-four hours later, diffuse bilateral atelectasis developed, in apparent concert with an increase in the minimum surface tension of saline lavage fluid. These changes were attributed to depletion, alteration, or interference with the activity of surfactant because of overinflation.[26] Barsch et al ventilated dogs for 1–3 days at PIP of 34 cm H_2O, and discovered, in addition to substantial atelectasis, vascular damage, pulmonary edema, hemorrhage, and hyaline membrane formation.[27]

Webb and Tierney showed that, in some preparations at least, pulmonary edema due to high-pressure mechanical ventilation could develop much more rapidly.[28] Normal rats were ventilated with a range of peak airway pressures. No pulmonary edema developed after 60 minutes at pressures of 14 cm H_2O, but moderate edema developed with 30 cm H_2O peak pressure. At pressures of 45 cm H_2O, severe pulmonary edema, hypoxemia, and decreased lung compliance were observed after as little as 13 minutes. Microscopic evaluation of the edema fluid showed an eosinophilic staining pattern consistent with a high protein content and suggestive of increased vascular permeability and exudation.[28] Electron microscopic observations in a similar model showed a pattern of diffuse alveolar damage and endothelial disruption.[29]

Kolobow et al explored the effects of high-pressure ventilation in healthy sheep[30] to determine whether distending pressures similar to those routinely used in the clinical management of acute respiratory failure were injurious to large animal lungs. They were motivated by a compelling disparity in the outcomes of two extracorporeal membrane oxygenation (ECMO) trials. The National Institutes of Health-sponsored ECMO collaborative study had failed to show a mortality benefit when ECMO was used as an adjunct to "state of the art" ventilatory management.[31] Gattinoni et al showed dramatically better results when similar patients placed on ECMO underwent low-frequency ventilation with PIP < 35 cm H_2O, an improvement they attributed to reduced ventilator-associated lung injury.[32] Consistent with that hypothesis, Kolobow et al found that mechanical ventilation of healthy paralyzed sheep at PIP of 50 cm H_2O led, over a matter of hours, to deteriorating pulmonary mechanics, respiratory failure, and severe lung damage at autopsy. By contrast, V_T of 10 mL/kg with associated PIP of 15–20 cm H_2O yielded preserved mechanics and largely normal autopsy findings. These investigators subsequently showed similar, though somewhat milder, deleterious effects in sheep ventilated with PIP of 30 cm H_2O.[33]

"Barotrauma" versus "Volutrauma"

Building on an experimental approach pioneered 50 years earlier,[34] Dreyfuss et al undertook to determine whether the insulting agent in studies of mechanical ventilation was in fact high airway pressure, as usually described, or consequent high volumes. As had been previously described in several studies, healthy rats ventilated with PIP of 45 cm H_2O and resultant V_T of 40 mL/kg developed enlarged, congested lungs with increased microvascular permeability as determined by radiolabeled protein leakage. Lung appearance and vascular permeability were preserved in rats ventilated with PIP of 7 cm H_2O and V_T of 13 mL/kg. Rat lungs ventilated with PIP of 45 cm H_2O but V_T constrained by thoracoabdominal banding to 19 mL/kg appeared normal by both light and electron microscopy, and maintained normal microvascular permeability. Finally, lungs of rats ventilated to V_T of 44 mL/kg with negative inspiratory pressure via an iron lung resembled those of the high-pressure/high-volume group.[35] Thus it appeared that "volutrauma" was a more apt description than the traditionally accepted "barotrauma".[36] Similar results were reported in other species, including rabbits[37] and lambs.[38]

Injured Lungs

In the studies reported above, high-pressure/high-volume ventilation was shown to lead to a pattern of diffuse alveolar damage in normal animal lungs. It is reasonable to argue that these results may be pertinent at least to the impact of mechanical ventilation on relatively spared lung regions in human ARDS. Additional studies have found that similar ventilatory strategies exacerbate preexisting injury in chemical models of ARDS. Bowton and Kong found higher lung weights in oleic acid-injured lungs exposed to high V_T (18 mL/kg) than in those exposed to low V_T (6 mL/kg).[39] The lack of uninjured, mechanically ventilated controls rendered their results difficult to interpret. This was rectified by Hernandez et al, who found that oleic acid injury decreased the pressure threshold for ventilator-induced injury relative to healthy lungs (Fig. 3).[40] The results of Dreyfuss et al were still more intriguing. Using α-naphthylthiourea (ANTU) to create moderate permeability edema, they showed that the injurious chemical effects were unaffected by low volume (7 mL/kg) ventilation. ANTU-associated and ventilation-associated injury were additive at intermediate V_T (25–33 mL/kg). At 45 mL/kg, however, lung damage was synergistic, exceeding the arith-

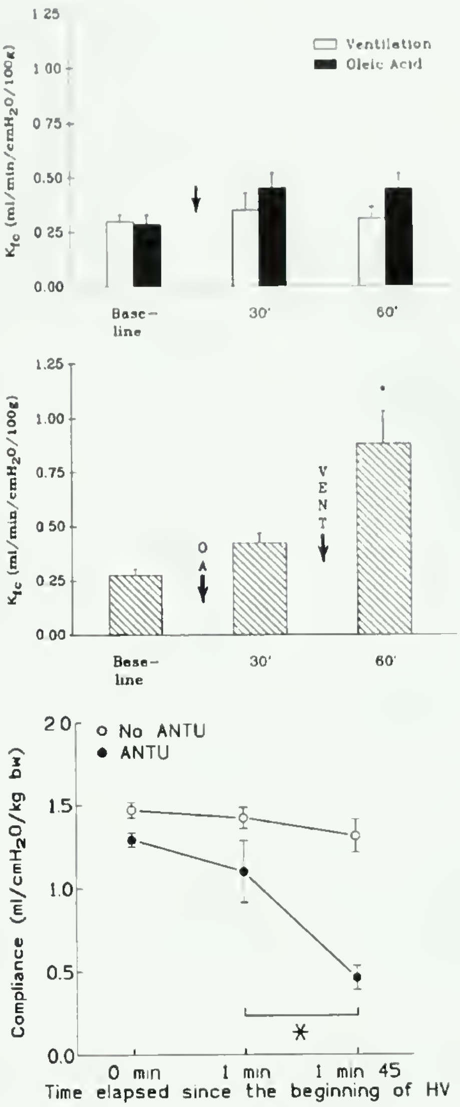

Fig. 3. Injured lung has heightened susceptibility to damage from mechanical ventilation. Upper graph: Comparison of capillary filtration coefficient (K_{fc}: a measure of endothelial integrity) in isolated rabbit lung at baseline and after oleic acid (OA, a chemical irritant) or mechanical ventilation with 25 cm H_2O peak inspiratory pressure. There is mild trend toward increased capillary leak with mechanical ventilation; slightly more with oleic acid injury. Middle graph: Injury from mechanical ventilation is significantly increased following oleic acid infusion. Lower graph: In intact rat lung, compliance decreases significantly with high-volume ventilation (HV: 45 mL/kg body weight) following chemical injury with α-naphthylthiourea (ANTU), compared with lungs without prior ANTU exposure. * = $p < 0.05$ for both studies. (Upper and middle graphs from Reference 40, with permission. Lower graph from Reference 41, with permission.)

metic sum of the expected chemical injury and volutrauma.[41]

Inflammatory Mediators

In addition to the direct mechanical effects of overinflation, VILI may involve a host of inflammatory mediators. Woo and Hedley-Whyte noted an accumulation of leukocytes and alveolar macrophages in the overinflated lungs of open-chested dogs.[42] Tsuno et al observed inflammatory cell infiltration in piglet lungs with diffuse alveolar damage induced by high-pressure mechanical ventilation.[43] Kawano et al demonstrated that saline-lavaged lungs of neutropenic rabbits did not develop increased permeability or hyaline membranes, unlike those of control rabbits.[44] Hyaline membranes were seen to develop in ventilated isolated rat lungs despite exsanguination,[45] but these lungs had been previously lavaged and may already have recruited neutrophils. More recently, Tremblay et al found that bronchoalveolar lavage fluid from isolated rat lungs after high-volume, zero PEEP ventilation had high levels of tumor necrosis factor α, interleukin 1β, interleukin 6, and macrophage inflammatory protein.[46] Ranieri et al found a similar profile in bronchoalveolar lavage fluid from ARDS patients undergoing traditional ventilation. This landmark study found a significant reduction in these inflammatory mediators with use of lung-protective ventilation.[47] Von Bethmann et al detected a similar profile in the perfusate of isolated mouse lungs ventilated at high volumes with negative pressure ventilation, suggesting that cytokines in an intact animal might be released into the systemic circulation.[48] Like the demonstration by Nahum et al that high V_T, low PEEP ventilation in dogs promoted translocation of bacteria from the lung into the general circulation,[49] systemic cytokines provide a plausible mechanism for the multiple organ system failure that is the most feared and lethal complication of ARDS.[50]

Low Tidal Volume Injury and Positive End-Expiratory Pressure

Though a preponderance of evidence suggested that mechanical ventilation incited lung injury through excessive pressures or volumes, two observations proved perplexing. The first was the inconsistent observation that PEEP could ameliorate the deleterious effects of high-pressure/high-volume ventilation. Webb and Tierney demonstrated that the protective effect of PEEP added to a high-pressure/high-volume ventilation strategy. Rats ventilated with 45 cm H_2O end-inspiratory pressure developed alveolar edema, hypoxia, and high lung weight, while those with the same end-inspiratory pressure but 10 cm H_2O PEEP had no edema and better gas exchange.[28] A similar effect of PEEP was observed by Corbridge et al in mechanically ventilated dogs after induction of diffuse alveolar damage with acid aspiration.[51] Both groups of investigators reasoned that low end-expiratory pressures and consequent alveolar hypoinflation led to surfactant inactivation and possible displacement into airways. Loss of surfactant activity would then increase alveolar surface tension and atelectasis, and perhaps also decrease perimicrovascular pressures, promoting fluid transudation.[28,51,52] The possibilities that higher PEEP might increase Zone I conditions in the lung, or promote lymphatic clearance, were also entertained. Dreyfuss et al found less interstitial and no alveolar edema in rats ventilated with high-pressure/high-volume with the addition of 10 cm H_2O PEEP, and made the important observation that epithelial and endothelial ultrastructure were preserved, arguing for a true "protective effect" of PEEP.[35] Since PIP was kept the same, V_T were smaller in the PEEP group (25 mL/kg) than in the group without PEEP (40 mL/kg). This raised the possibility that it was the size of the ventilatory excursion itself that was injurious. However, rats undergoing normal V_T ventilation from high functional residual capacity during negative pressure ventilation had gross and ultrastructural lung damage similar to that of the high-pressure/high-volume group, suggesting it was total lung distention rather than large tidal excursions per se that were injurious.[53] As the authors note, whatever its source, "any form of lung overinflation is noxious."[36]

The second perplexing observation was that mechanical ventilation of injured lungs with low V_T could also exacerbate lung injury. In normal lungs, small airways and alveolar units do not collapse at end expiration, and relatively low V_T are usually not injurious. In lungs with ARDS-like injury, regional collapse occurs at end expiration. During normal tidal breathing, some areas can be observed to undergo cyclic opening and collapse.[9] Possible implications of this for surfactant activity are discussed above. Following upon the work of Mead et al.[22] Robertson hypothesized that repeated opening and closing might also generate shear stresses that would exacerbate lung injury.[54] Collapsed airways would have a threshold opening pressure. When that pressure was exceeded during a tidal breath, the airway would open progressively from the proximal to the distal portion. The pressure wave moving along the airway would apply tangential pressure to the epithelium, creating shear stress. Wall tension at the junctions between open and collapsed alveoli might be substantially higher than end-inspiratory pressure.[22] Low V_T in injured lungs could therefore cause progressive injury, while application of PEEP sufficient to maintain airway patency at end-expiration should be protective. This was tested in a rabbit model of ARDS by Sandhar et al, who found less injury in saline-lavaged lungs with PEEP sufficient to maintain airway patency than with lower levels of PEEP.[55] To circumvent the confounding effects of different pulmonary blood flows and blood gases with different ventilatory strategies. Muscedere et al performed a similar experiment in lavaged, explanted rat lungs.[45] Lungs ventilated for 2 hours with 5–6 mL/kg V_T and sufficient PEEP to maintain open lung units had injury scores similar to control lungs exposed to a static inflation pressure of 4 cm H_2O. Lungs ventilated with similar V_T but lower levels of PEEP had more severe damage. Strikingly, the location of injury varied with the PEEP level, moving more proximally as PEEP was reduced. Thus, two important problems vexing proponents of a high-pressure/high-volume mechanism of VILI appeared to have an important conceptual overlap. Low V_T could be injurious if they involved pressure swings that crossed the opening pressures for recruitable lung units in damaged lungs. PEEP, though potentially increasing the risk of overdistention, could be protective if it maintained end-expiratory pressures above closing pressures for those units.

Concerns over both overdistention and cyclic recruitment and derecruitment of alveolar units have led many experts to suggest the utility of pressure-volume (P-V) curves in individualizing ventilatory parameters. A P-V curve of the respiratory system is generated with use of a large-volume syringe or, more typically, by using the ventilator to evaluate static compliance through a graded series of inflations. In many ARDS patients the P-V curve assumes a sigmoidal shape (Fig. 4). The lower point of maximum curvature is usually referred to as the lower inflection point or P_{flex}. The upper point of maximum curvature is usually referred to as the upper inflection point (UIP). While there are various interpretations, the lower inflection point is commonly thought to be caused by the inflation of large numbers of recruitable lung units and the decreased compliance above UIP to suggest increased risk of overdistention. On this view, PEEP should be set above the lower inflection point and V_T adjusted to keep plateau pressures below the UIP. The P-V curve is theoretically attractive, and has guided ventilator adjustments in several clinical trials.[56–58] There remains intense debate, however, over the relative utility of inflation and deflation curves, the significance of the "inflection" points, and the best application of the P-V curve to the adjustment of ventilatory parameters. Its clinical utility remains unproved. A more detailed discussion of the vast literature and myriad subtleties of PEEP, P-V curves, and alveolar recruitment is beyond the scope of this article.

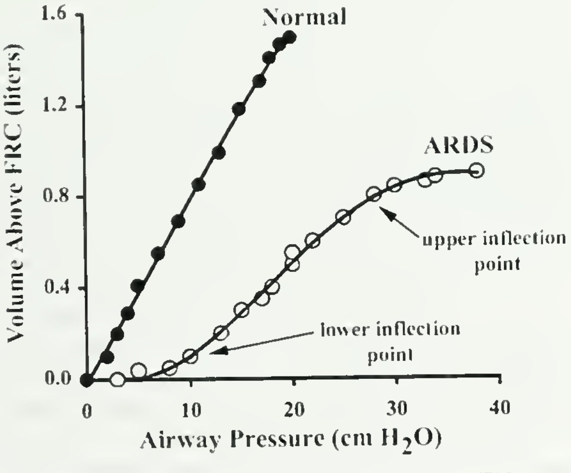

Fig. 4. Idealized respiratory system pressure-volume (PV) curve of a normal subject and of an ARDS patient. The ARDS curve has a lower slope, reflecting decreased compliance, a "lower inflection point," reflecting a pressure at which substantial recruitment may occur, and an "upper inflection point," above which the risk of overdistention may increase. FRC = functional residual capacity. (Adapted from Bigatello LM, Hurford WE, Pesenti A. Ventilatory management of severe acute respiratory failure for Y2K. Anesthesiology 1999;91(6):1567–1570.)

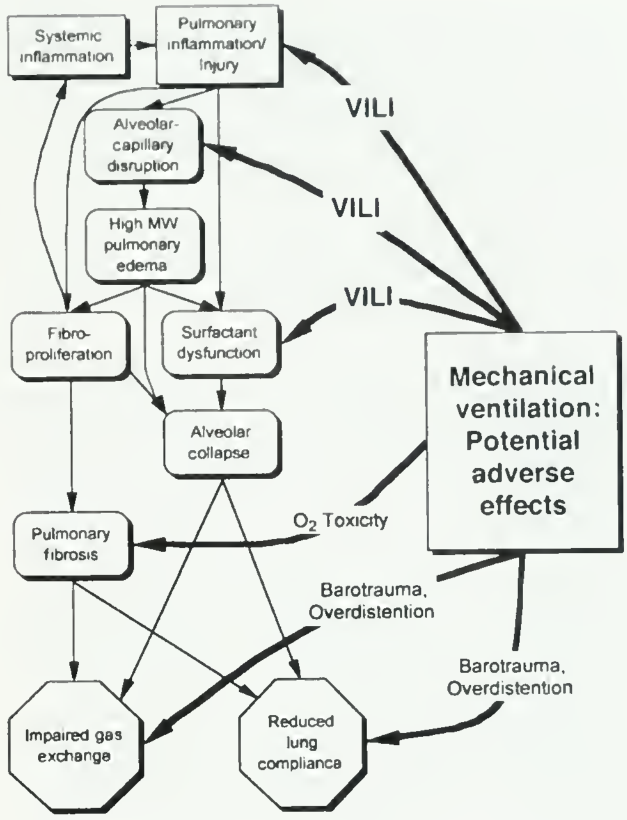

Fig. 5. Ventilator-induced lung injury (VILI) may exacerbate damage from acute respiratory distress syndrome (ARDS). Small boxes and light arrows provide a hypothetical schema for ARDS pathophysiology. Large box and bold arrows represent possible contributory effects of mechanical ventilation. MW = molecular weight. (From Reference 12, with permission.)

Collectively, there is extensive support for the idea that ventilation at both high and low lung volumes, at either extreme of the compliance curve of the damaged lung, can worsen pulmonary mechanics, gas exchange, and lung injury (Fig. 5). Injured but recruitable lung demands sufficient end-expiratory pressure to open and to remain open. This static pressure may be sufficient to substantially distend a region of uninjured lung, moving it near to a compliance plateau. Even small additional volume increments from tidal breathing may then be enough to cause overdistention injury.

Contemporary Mechanical Ventilation in Acute Respiratory Distress Syndrome: Lung Protection and Permissive Hypercapnia

The practical challenges posed by the potential for mechanical ventilation to exacerbate lung injury, together with the theoretical difficulty of establishing the parameters of a truly benign ventilatory strategy, promoted alternative gas exchange strategies that avoided large volume excursions and "put the lung at rest."[36] One such approach is the use of high-frequency oscillation, a common ventilatory modality in management of hyaline membrane disease of neonates. Although it has been the subject of close study for decades, the pathophysiologic,[59] morbidity, and mortality benefits of high-frequency oscillation have not been conclusively demonstrated.[60,61] A second approach is the use of intravenous[62,63] or extracorporeal gas exchange devices[31,32] to partially or completely assume the roles of oxygen and carbon dioxide (CO_2) transfer. While these approaches are technically feasible and have proponents, they are highly invasive, expensive, available only in major referral centers, and subject to substantial complications, and their benefits remain unproved in adults.[31,32,62,64] An alternative that has emerged as the standard of contemporary ventilator management in ARDS is to strictly limit V_T, tolerating marked alveolar hypoventilation and the consequent hypercapnia.

Although the benefits in status asthmaticus had been previously defined,[65] Hickling et al brought the concepts of diligently limited PIP and "permissive hypercapnia" to the clinical forefront of ARDS management.[66] They retrospectively reviewed records of 50 ARDS patients whose ventilatory support was constrained to PIP $< 30-40$ cm H_2O by reductions in V_T and by "disregarding hypercapnia." Though V_T were "sometimes as low as 5 mL/kg," minimal V_T was more typically 8–10 mL/kg. Mean and maximum measured P_{aCO_2} were 62 mm Hg and 129 mm Hg, respectively, with concordant pH of 7.23 and 7.02, and substantial base excesses. While there were no controls, mortality was much lower than predicted on the basis of Acute Physiology and Chronic Health Evaluation I scores, lung injury scores, and ventilator scores.

Although Hickling's report was provocative and compelling, adoption of pressure-limited ventilation and permissive hypercapnia as standards of care depended critically on the answers to two questions. The first was whether hypercapnia could indeed be merely "disregarded" in the management of critically ill patients. The second was whether the benefit of such a lung-protective strategy would prove robust in prospective, controlled clinical trials.

Physiologic Consequences of Permissive Hypercapnia

Since the theoretical and apparent clinical benefits of a lung-protective strategy incorporating permissive hypercapnia were first put forward for ARDS, considerable attention has focused on the effects of hypercapnia and respiratory acidosis at the cellular and systemic levels, in animals, anesthetized humans, and patients with respiratory failure. Though much remains to be learned, the broad outlines seem clear: the effects of hypercapnia are myriad, complex, subtly interwoven, and in general remarkably well tolerated by even the critically ill.

Healthy humans and most other terrestrial mammals assiduously defend a P_{aCO_2} near 40 mm Hg, inviting teleological arguments that to disturb that value is to place the organism at peril. Though at 37° C a blood pH of 7.40 may promote an intracellular pH ideal for protein ionization,[67] this blood pH could be maintained over a wide range of P_{aCO_2}. It has been elegantly proposed that a P_{aCO_2} of 40 mm Hg strikes an optimum balance between respiratory work and arterial oxygenation.[68] If P_{aCO_2} rises, P_{aO_2} falls, by the alveolar gas equation; at a P_{aCO_2} near 100 mm Hg, hypoxemia may be life-threatening. Decreases in P_{aCO_2} below 40 mm Hg do little to augment P_{aO_2}, at substantial metabolic cost for increased ventilation. The optimization argument assumes, however, that F_{IO_2} is constrained to 0.21. With supplemental oxygenation, a much higher P_{aCO_2} can be tolerated without threatening hypoxemia.

A natural extension of the above argument is that circumstances may prevail in which the optimum "set point" for P_{aCO_2} is considerably higher (Fig. 6). When impaired respiratory mechanics make the metabolic cost of maintaining sufficient alveolar ventilation to defend a P_{aCO_2} of 40 mm Hg prohibitively high, there may be a useful economy in allowing CO_2 to accumulate to a new steady state. This is the presumed adaptation behind the chronic hypercapnia of many patients with severe obstructive lung disease and the argument against ventilating such patients to eucapnia during exacerbations requiring mechanical support. Similar advantages accrue to the strategy in ARDS, wherein a large dead space-to-V_T ratio impairs the efficiency of ventilation[70] and hypermetabolism can lead to important increases in CO_2 production.[71] Furthermore, the concomitant rightward shift of the oxyhemoglobin dissociation curve in the setting of respiratory acidosis (the Bohr effect) favors oxygen unloading in the periphery, which may be an optimal approach toward the different goal of oxygen delivery to a tissue in crisis.

The broader functional and physiologic consequences of permissive hypercapnia have been well reviewed.[72] CO_2

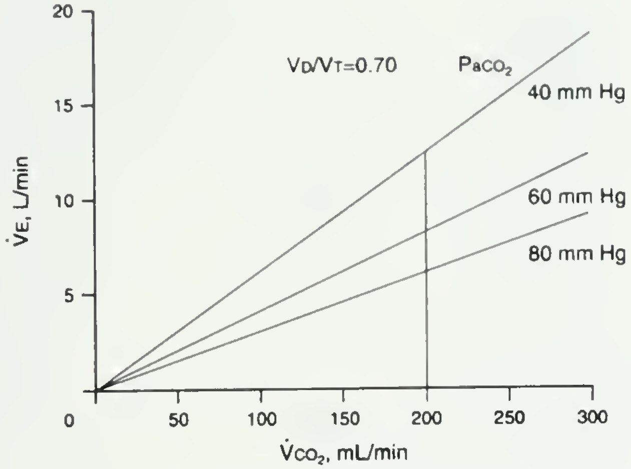

Fig. 6. Permissive hypercapnia decreases ventilatory demand. Estimated minute ventilation ($\dot{V}_E$) requirement as a function of CO_2 production ($\dot{V}_{CO_2}$) for 3 arterial partial pressure of carbon dioxide (P_{aCO_2}) levels, assuming dead space fraction (ratio of dead space volume to tidal volume, V_D/V_T) of 0.7. Compared with P_{aCO_2} of 40 mm Hg, minute ventilation is 34% lower at 60 mm Hg and 50% lower at 80 mm Hg. Vertical line indicates normal CO_2 production. (From Reference 69, with permission.)

accumulating in blood diffuses freely across erythrocyte and other cellular membranes, and is converted by cytosolic carbonic anhydrase to carbonic acid, which rapidly ionizes. The consequent intracellular acidosis is checked by immediate, partial physicochemical buffering; intracellular pH is typically restored to normal over 1–3 hours by additional mechanisms, including transmembrane H^+ and HCO_3^- flux.[69] These potent regulatory mechanisms essentially preclude hypercapnia from generating severe intracellular acidosis. The transient acidosis that develops, however, can cause many disturbances.[72] Glycolysis is decreased, albeit with a well-preserved cellular energy state. Abnormal excitation-contraction coupling and actin-myosin binding lead to malfunction of contractile elements. Cell membrane ion conductances and electrolyte transport are modulated, altering the properties of excitable cells. These changes are thought to be reversible with correction of intracellular acidosis.

Though the intracellular effects of a discrete CO_2 challenge are rapidly neutralized, full metabolic compensation for a systemic respiratory acidosis via renal acidification requires 1–5 days.[73,74] Despite the potentially ubiquitous effects of this sustained acidosis, however, substantive effects appear to be limited to the cardiovascular and central nervous systems (CNS). In general, these systemic consequences comprise a synthesis of local tissue effects and global sympathetic activation.[75]

Hypercapnia has been known for almost a century to depress myocardial contractility in isolated hearts.[76] This effect is completely reversible.[77] In intact animals, however, the reduction in myocardial contractility is more than offset by the stimulatory effects of hypercapnia on the sympathetic nervous system (Fig. 7).[78,79] Catecholamine release increases heart rate and may increase stroke volume, leading to a net increase in cardiac output[79,80] (Fig. 8). This is probably adaptive in many circumstances, but may place unacceptable demand on a failing or ischemic heart.[81] Furthermore, though CO_2 is a coronary vasodilator, its net effect on perfusion of ischemic myocardium is unclear.[71]

CO_2 causes peripheral vascular smooth muscle relaxation,[82] an effect again opposed by sympathetic vasoconstriction. The consequence is usually a mild net decrease in systemic vascular resistance (SVR).[71,79] Venous capacitance is also reduced.[83] Since preload is augmented and afterload reduced, cardiac output tends to be further enhanced. The slight drop in SVR is well compensated by the increase in cardiac output, leading to maintenance or, more often, increase of mean arterial blood pressure. This can be compromised by pharmacologic β blockade.[80,84]

Though systemic vascular resistance decreases with respiratory acidosis, pulmonary vascular resistance is maintained or increased because of vasoconstrictive effects and possible potentiation of hypoxic vasoconstriction. In the face of increased cardiac output, pulmonary arterial pressure rises and substantial pulmonary hypertension can develop.

Given the manifest vasoactivity of acute hypercapnia, its effects on local and regional blood flow are of great importance to its application in critically ill patients. Unfortunately, results differ between animals, preparations, and organs, and in particular are mixed for both splanchnic and renal perfusion.[71,72,85] Regional blood flow in a particular case cannot be predicted with confidence.

Moderate respiratory acidosis can lead to depressed consciousness, increased respiratory drive, and a lowered seizure threshold;[72] at high levels, CO_2 is anesthetic.[74] Importantly to patient care, pharmacological modulation of the increased respiratory drive usually requires high levels of sedation, and often neuromuscular blockade,[57,71,72,86] with the attendant possibilities of important neuromuscular sequelae.[87–90] The respiratory drive at any P_{aCO_2} can be suppressed by correction of the attendant acidosis.[91] Among the many CNS alterations due to permissive hypercapnia, however, cerebral blood flow and intracranial pressure (ICP) effects are the most profound. Because of both cerebral vasodilation and increased systemic blood pressure,[71] cerebral blood flow rises dramatically with P_{aCO_2}, plateauing at levels of P_{aCO_2} in excess of 100 mm Hg.[72] While there is some debate over the direct role of molecular CO_2,[72] it is generally thought that extracellular pH is the critical variable.[71,92] ICPs rise monotonically with flow, and can be several times normal. This degree of intracranial hypertension is generally associated with only modest neurological sequelae. In pathologic settings in which ICP

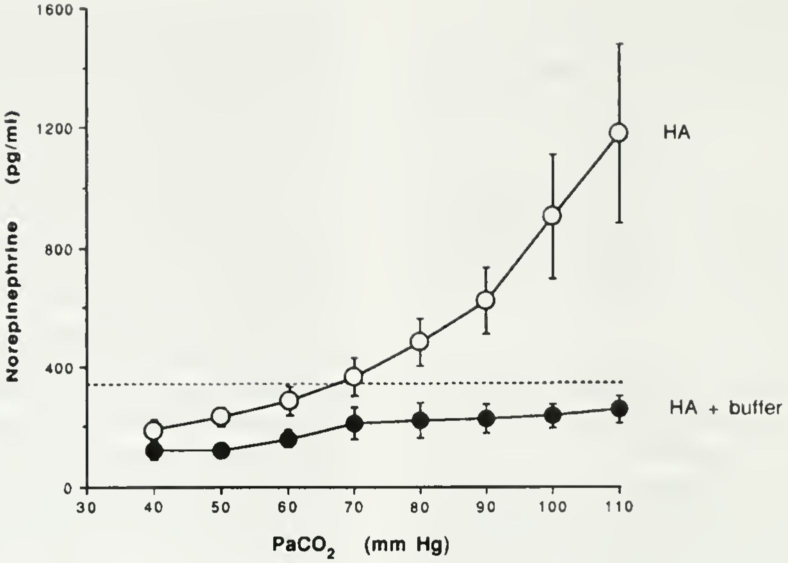

Fig. 7. Norepinephrine secretion in swine is markedly increased in acute hypercapnic acidosis (HA). Aggressive bicarbonate buffering (buffer) attenuates this effect. Other catecholamines are similarly increased (not shown). Dashed line = upper limit of basal plasma norepinephrine concentration. Data are mean ± SEM. (From Reference 79, with permission.)

or soft tissue volume are already increased, such as with cerebral edema, mass lesions, or head trauma, substantial respiratory acidosis can lead to mass effect, compromised perfusion, and even herniation, and should usually be avoided. It is notable, however, that while prophylactic hyperventilation has been recommended in acute head injury, its prolonged use has been associated with a worsened neurological outcome in subsets of patients with traumatic brain injury.[93]

It is widely held that it is the associated respiratory acidosis, rather than hypercapnia per se, that engenders the described profile of hemodynamic and vascular alterations. Consistent with this, hemodynamic and CNS effects can be markedly attenuated if P_{aCO_2} elevation is separated from the accompanying acidosis by the administration of base.[79,85] In sheep made acutely hypercapnic to a P_{aCO_2} of 80 mm Hg and pH of 7.14, cardiac output and carotid arterial blood flow increased markedly from baseline, and ICP rose 300%. When arterial pH was maintained in normal range by concomitant bicarbonate administration, cardiac output and carotid blood flow were significantly elevated only at the 1-hour time point, and ICP remained at baseline.[85] In swine, catecholamine secretion, decreases in SVR, and increases in cardiac output and heart rate were also attenuated, though not eliminated, by extracellular pH buffering with bicarbonate (see Fig. 7).[79] While the ability to blunt the systemic effects of hypercapnia is important, the delayed and incomplete nature of the attenuation is also of interest. Though blood pH was closely regulated, extracellular pH monitoring may not reflect intracellular conditions. Since biological membranes are much more permeable to CO_2 than to bicarbonate, it is unlikely that even bolus administration of bicarbonate prevents at least transient intracellular acidification. The prevention of intracranial hypertension, at least by the first measurement at 1 hour, supports the notion that cerebral blood volume is particularly sensitive to extracellular pH.

The depths of human tolerance to respiratory acidosis have been sounded on occasion, usually by iatrogenesis. A patient accidentally administered CO_2 during general anesthesia attained a P_{aCO_2} of 248 mm Hg and a pH of 6.86, but recovered without complication.[94] A patient undergoing cosmetic surgery, with adequate oxygenation but inadequate ventilation for over 4 hours, reached a P_{aCO_2} of 375 mm Hg and a pH of 6.6; despite postanesthetic coma there was an uneventful recovery.[95] Similarly severe respiratory acidosis has been tolerated by children for up to 2 days.[96]

Although the respiratory acidosis consequent to permissive hypercapnia is usually discussed in terms of its potentially deleterious effects, the argument has been advanced that acidosis may instead be adaptive and protective.[97] Augmentation of cardiac output and mean arterial pressure, together with a rightward shift in the oxyhemoglobin dissociation curve, can in theory promote oxygen delivery and unloading in tissues, though the uncertainty surrounding individual organ perfusion is described above. Decreased cellular respiration[71] may in turn decrease oxygen consumption. There are animal data suggesting that hypercapnic acidosis (specifically) may di-

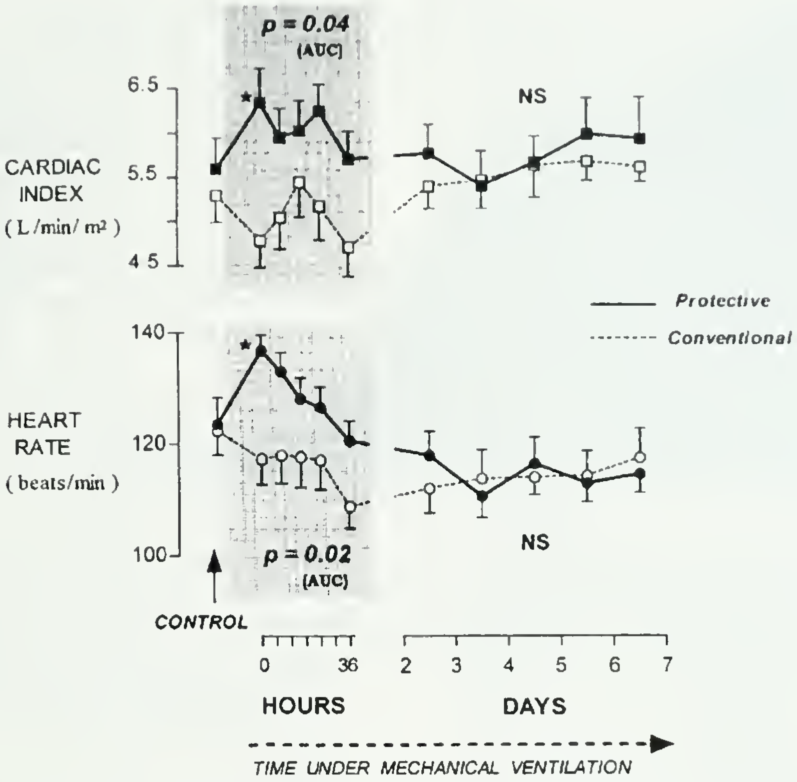

Fig. 8. Temporal evolution of cardiac index and heart rate in patients undergoing permissive hypercapnia. Increased cardiac index and heart rate are mediated by increased sympathetic tone due to respiratory acidosis. With metabolic compensation of acidosis, cardiac effects are normalized. AUC = incremental area under the curve. NS = not significant. (From Reference 57, with permission.)

minish ischemic reperfusion injury in heart, brain, and lung.[97] Acidosis also downregulates a host of inflammatory processes associated with tissue injury, decreases oxygen free-radical production in brain homogenates, and increases nitric oxide production in the lung.[97,98] Though the concept that respiratory acidosis is protective has expanding scientific grounding and has gained some theoretical credence among intensivists, much needs to be learned before the therapeutic possibilities for patient care can be seriously considered.

Taken together, these data suggest that acute respiratory acidosis is likely to be tolerated in most clinical settings, even by critically ill patients. Neither certainly safe nor certainly hazardous levels have been clearly defined, and commonly cited lower levels of clinician comfort in the pH range of 7.15 to 7.25 appear largely arbitrary. Known cardiovascular and CNS effects may be less well tolerated by certain patient subsets, and these subsets are in general the least well studied. Permissive hypercapnia is thus probably still best used circumspectly in patients with cardiac ischemia and left ventricular compromise, and with particular caution in patients with pulmonary arterial hypertension and right heart failure. Because of the potent effects of respiratory acidosis on cerebral perfusion and the potentially catastrophic consequences of increased ICP in susceptible patients, permissive hypercapnia is contraindicated in patients with serious head trauma or certain CNS pathologies. If clinical circumstances mandate its use, ICP monitoring is indicated. Concerns regarding respiratory acidosis are amplified in the setting of concurrent metabolic acidosis. As further data accrue from clinical applications of permissive hypercapnia (see below), these guidelines may be refined.

Clinical Consequences of Permissive Hypercapnia

As permissive hypercapnia in lung-protective mechanical ventilatory strategies has become more widespread, especially for the management of ARDS, a substantial amount has been learned about its implications for clinical practice. In general, these results accord well with those from the studies of animals and anesthetized humans described above.

Intracranial pathology is generally considered an absolute contraindication to permissive hypercapnia, limiting clinical data on its consequences in that setting largely to case reports. Tasker and Peters described a young patient with meningococcemia, meningitis, cerebral edema, and ARDS, and in whom electroencephalography and jugular oxygen saturation monitoring were used as an indirect gauge of cerebral oxygenation and hyperemia.[99] By those measures, clinically important elevations in ICP occurred below a pH of 7.32. With a slow, controlled increase in hypercapnia and progressive renal compensation, the authors were able to reduce PIP from 42 cm H_2O to 32 cm H_2O and V_T from 12 mL/kg to 6–8 mL/kg over 24 hours.

The hemodynamic effects of permissive hypercapnia in ARDS patients without CNS pathology or severe metabolic acidosis have been studied in several series. Thorens et al induced hypercapnia by decreasing V_T over 30–60 minutes in 11 ARDS patients and studied its effects over 2 hours.[86] Mean P_{aCO_2} of 59 mm Hg and pH of 7.26 were attained, with a 40% reduction in minute ventilation and a small but significant decrease in mean PIP, from 32.5 cm H_2O to 30.6 cm H_2O. Surprisingly, heart rate remained unchanged; cardiac index increased by 18% and SVR decreased by 25%, with a net small decrease in mean arterial pressure that approached significance. PVR did not change, but mean pulmonary artery pressure increased significantly, from 29 mm Hg to 32 mm Hg. Notably, while left ventricular stroke work was unchanged, right ventricular stroke work increased by 25%. Venous admixture was increased; increased F_{IO_2} was required to compensate P_{aO_2} reductions in a subset of patients. Oxygen delivery increased, but consumption was unaltered. Carvalho et al compared hemodynamics over time between 23 traditionally managed ARDS patients and 25 ARDS patients managed with a lung-protective strategy characterized by $V_T < 6$ mL/kg and PEEP set above the lower inflection of the static pressure-volume curve.[57] In the lung-protective group, average and absolute maximum P_{aCO_2} were 70 mm Hg and 114 mm Hg, respectively; average and absolute minimum pH were 7.13 and 6.88. Slow bicarbonate infusions were administered for $pH < 7.2$. In patients undergoing permissive hypercapnia, there were highly significant immediate increases in heart rate, cardiac output, and oxygen delivery, with a decrease in SVR and no change in oxygen consumption (see Fig. 8). Consonant with Thorens's results, PVR was unchanged but mean pulmonary artery pressure increased. Statistical analyses suggested that all alterations were attributable to respiratory acidosis rather than to elevated PEEP. Notably, all acute hemodynamic effects were progressively attenuated over 36 hours despite maintained hypercapnia, in concert with renal compensation of the acidosis. In both of the above series, acute hypercapnia was generally well tolerated, without important adverse effects that could be attributed to its hemodynamic or other consequences. Both sets of authors noted, however, that their results reinforce caution in its use in patients with overt heart failure, severe pulmonary hypertension and/or right ventricular dysfunction, and severe hypoxemia.[57,86] Although not specifically designed to evaluate the hemodynamic or other systemic consequences of respiratory acidosis, numerous other studies have failed to show obvious adverse effects from permissive hypercapnia in ARDS patients.[58,100,101] Its safety, however, remains at issue, as described below.

Mortality Benefits of Low Tidal Volume Ventilation and Permissive Hypercapnia in Acute Respiratory Distress Syndrome

The strong theoretical and experimental bases for lung-protective ventilatory strategies, coupled with encouraging preliminary clinical results, inspired a number of efforts to prove their merit in controlled clinical trials. Initial Canadian[100] and European[102] multicenter trials did not demonstrate a mortality benefit from limitations on alveolar pressure and V_T; the former suggested possibly increased morbidity. Several features of study design and implementation may have contributed to these outcomes. First, with 120 and 116 patients, respectively, neither was powered to detect small differences in outcome. Second, differences in V_T between intervention and control groups, though significant, were not large, being 7.2 mL/kg versus 10.8 mL/kg ideal body weight in the Canadian study, and 7.1 mL/kg versus 10.3 mL/kg dry body weight in the European study.[102] Though both groups of authors suggested that a mortality difference did not emerge because the control ventilatory strategy was itself protective,[100,102] it is at least as likely that limits in the intervention group were insufficiently rigorous.[101] Indeed, investigators in the European study had previously reported on the extent of V_T and ventilatory pressure limitations necessary to keep plateau pressures under the measured UIP of the pressure-volume curve in 25 ARDS patients (Fig. 9). Even at V_T of 7.1 mL/kg (the lower range attained in the randomized trial), approximately 20% of patients would have had plateau pressures above UIP,[56] supporting the possibility of sufficiently common overdistention injury to dampen any signal of the benefit of low V_T. Even when V_T kept plateau pressures below UIP, overdistention may have occurred; the authors reported a 12.7% rate of pneumothorax.[56] This effect might have been amplified by important differences in the way PEEP was set between the preliminary and randomized trials. Finally, and of particular interest in the present context, both sets of investigators, and others, have raised the possibility that mortality benefits due to reduced V_T were negated by deleterious effects of controlled hypoventilation, including inadequate alveolar recruitment[102] and particularly respiratory acidosis.[100,103] In the European study, mean P_{aCO_2} at day 1 of intervention was 59.5 mm Hg in the treatment group and 41.3 mm Hg in the control group. Corresponding values of pH are not

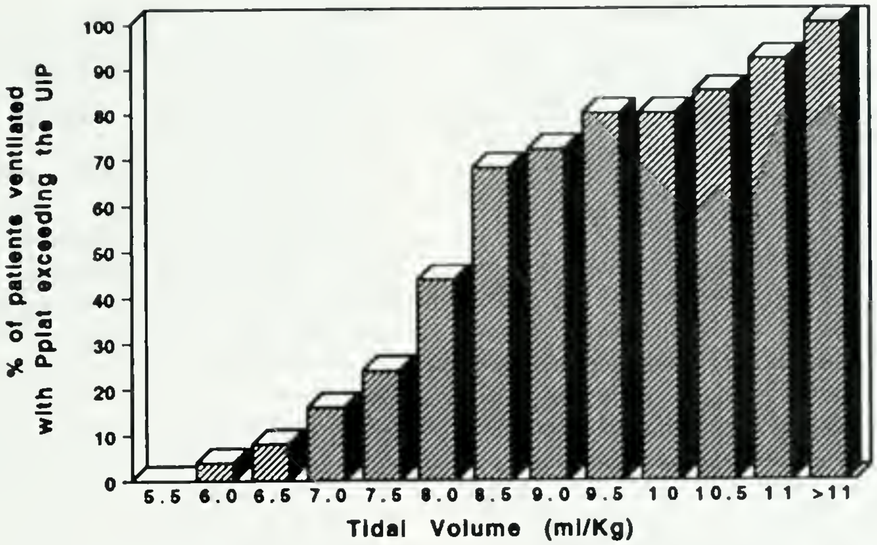

Fig. 9. Risk of overdistention injury in acute respiratory distress syndrome (ARDS) patients can occur at low tidal volume. Plot summarizes the percentage of ARDS patients in whom plateau pressure would exceed the upper inflection point (UIP) of the respiratory system pressure-volume curve as a function of tidal volume during mechanical ventilation. Positive end-expiratory pressure was adjusted to the lower inflection point. P_{plat} = plateau pressure. (From Reference 56, with permission.)

reported, but no correction of acidosis was employed unless pH fell below 7.05.[102] Smaller mean differences were observed in the Canadian study, but no attempts were made to correct respiratory acidosis unless pH fell below 7.0.[100] It is notable that neuromuscular blocking agents were required significantly more frequently in cases than in controls in the Canadian study; a similar trend in the European Study did not attain significance.

Amato et al found a significant reduction in 28-day mortality in ARDS patients mechanically ventilated with a lung-protective strategy, compared with traditionally managed controls.[58] PEEP levels, ventilatory modes, and V_T all differed between the groups, preventing assessment of the relative importance of any single intervention. Survival to hospital discharge was not different between the groups. Mean P_{aCO_2} and pH were significantly different over the first 36 hours, being 55–58 mm Hg and 7.19–7.25 for the intervention group and 33–35 mm Hg and 7.37–7.40 for the control group, respectively. The difference in P_{aCO_2} was sustained, but the difference in pH disappeared at later time points. Slow bicarbonate infusions were begun for pH < 7.2. Significantly more sedative and paralytic agents were required for the intervention group.[58] The study has been criticized, particularly for the unusually high 71% mortality of the control group.

Doubts over the ability of protective ventilatory strategies to reduce ARDS mortality were laid to rest with the recent publication of the ARDS Network trial. Lower mortality (39.8% vs 31%) was seen in patients supported with mean V_T of 6.2 mL/kg than in those supported with mean V_T of 11.8 mL/kg ideal body weight.[101] It is notable that mean plateau pressures, thought to best reflect alveolar distending pressures, were 25 cm H_2O and 33 cm H_2O in the intervention and control groups, respectively, remarkably similar to those of 25.7 cm H_2O and 31.7 cm H_2O in the European trial[102] and to mean PIPs of 23.6 cm H_2O and 34 cm H_2O in the Canadian trial.[100] The ARDS Network protocol specified a pH goal of 7.3 to 7.45 for both traditional and low V_T groups. This was achieved in the latter by significantly higher respiratory rates, supplemented by bicarbonate infusions as required. There was no significant difference between groups in the amount of neuromuscular blocking agent required.[101]

Though proof of a mortality benefit to a low V_T ventilatory strategy in ARDS represents a major advance, the critical variable in that benefit remains the subject of some debate. It is quite plausible that it is the intended intervention, low V_T, itself. Given the high respiratory rates of patients in the intervention group and the possibility of intrinsic PEEP, it is possible that the benefit was at least partly a derivative PEEP effect. The importance of PEEP in ARDS management is the focus of a new National Institutes of Health ARDS Network trial.[104]

Respiratory Management in Acute Respiratory Distress Syndrome: A Role for Tracheal Gas Insufflation?

The extraordinary research efforts into the nature and mechanisms of VILI, the pathophysiology of ARDS, and the physiology of hypercapnic acidosis have led to more sophisticated, and now more effective, patient management. Abundant room for improvement remains. A brief synopsis of the foregoing review highlights a subset of the important open questions:

1. It has been shown beyond reasonable doubt that overdistention and cyclic inflation and deflation of injured lung can cause further lung injury. Although this appears to take longer in larger animals than in smaller animals, there are no compelling data to suggest a safe interval for a nonprotective ventilatory strategy in humans. Increasing data suggest that VILI-associated generation of inflammatory mediators may contribute to a systemic inflammatory response syndrome and multiple organ system failure, the leading cause of death in ARDS.

2. Low V_T ventilatory strategies confer a proven mortality benefit in ARDS, probably by reducing VILI. The precise mechanism conferring that benefit remains somewhat uncertain.

3. Consequences of permissive hypercapnia have been studied from the cellular level to the systemic physiology of ARDS patients. Effects of the attendant respiratory acidosis are very widespread and certainly not fully delineated. The intriguing and important possibility that this acidosis may be adaptive or protective in some settings is gathering support but remains largely conjectural. The gross systemic effects of hypercapnic acidosis, vasodilation, and isolated myocardial depression compensated by catecholamine-induced partial vasoconstriction and a hyperdynamic state appear to be generally well tolerated by animals, anesthetized humans, and critically ill patients. However, certain patient subsets, particularly those with ischemic heart disease, left ventricular compromise, pulmonary hypertension, right heart failure, head trauma, intracranial disease, or coexistent metabolic acidosis, may be at higher risk. Furthermore, controlled trials in which a mortality benefit of lung-protective ventilation was apparent used an increased respiratory rate[101] and/or bicarbonate infusion[58,101] to control acidosis. Finally, acidosis is associated with dyspnea and increased respiratory drive and is the likely culprit in the increased use of sedatives and paralytics reported in many clinical trials of permissive hypercapnia. Limiting acidosis may enhance patient comfort and reduce the manifold important complications associated with administration of those pharmacologic agents.

The first and second elements of this synopsis suggest that a lung-protective strategy must *always* be considered a priority (though not necessarily the defining priority) in the management of the ARDS patient. They support the position of Carvalho et al that "except for situations of previous heart and/or neurological disease . . . the installation of [permissive hypercapnia] in a progressive and gradual manner is not justifiable and may consist in a risky waste of time".[57] The third element, however, supports the position of most experts that permissive hypercapnia is important but should be introduced gradually, and may need to be avoided altogether in susceptible populations.[71,72,104–107] How can these well-reasoned but disparate conclusions be reconciled?

An obvious approach is the immediate implementation of a lung-protective, low V_T ventilatory strategy in a patient with ARDS, but with close control of acidosis pending renal compensation. This can be accomplished, broadly, in two ways, not mutually exclusive: by allowing P_{aCO_2} to rise rapidly, but neutralizing the acidosis, or by controlling the rise of CO_2. The first of these can be achieved with supplemental buffering. Bicarbonate is inexpensive and widely available, and has been shown to attain the desired effect in some animal models.[79,85] In addition to unproved clinical benefits, however, concerns over bicarbonate abound: because of urinary excretion it is inefficient;[71] it may be ineffective;[108] it represents a substantial sodium load; it generates CO_2 that will need to be cleared precisely in a setting in which ventilation is at issue;[108,109] and if rapidly administered it has been found, at least in the setting of metabolic acidosis, to cause "paradoxical" intracellular acidosis.[110] Carbicarb is an effective alternative that does not generate CO_2[71] and avoids the transient intracellular acidosis of rapidly administered bicarbonate,[111] but is relatively unproved clinically. Tris-hydroxymethyl aminomethane has a greater buffering capacity than bicarbonate, does not elevate[109] and may reduce[108] CO_2, and generates bicarbonate. It has recently been recommended for use in acute lung injury,[108] though there have been longstanding concerns about possibly deleterious arterial vasodilatory effects,[112] and its renal excretion limits its usefulness in patients with renal insufficiency.

Setting aside invasive and technically intensive interventions such as extracorporeal CO_2 removal, control of the rate of CO_2 rise can be achieved by decreasing its production or increasing the efficiency of its elimination. There are close boundaries to what can be done to limit production: control of fever, avoidance of overfeeding, and neuromuscular paralysis are among the approaches commonly employed. A very promising means of improving ventilatory efficiency involves flushing the anatomic dead space through the technique of TGI.[113–115] Control of acidosis by augmentation of CO_2 clearance affords numerous potential advantages over other approaches. It can be initiated contemporaneously with adoption of a lung-protective ventilatory strategy, and its effects should be "instantaneous." Because it checks hypercapnia itself, it should preclude even transient intracellular acidosis, unlike the currently favored approach of bicarbonate administration. It may improve alveolar oxygenation. It does not represent an intravascular volume load. It is arguable that TGI should not be used to *eliminate* CO_2 accumulation but to *control* its rate to allow for renal compensation; maintenance of steady state at a higher P_{aCO_2} will always require less alveolar ventilation. It is important to remember in this connection that TGI affords not only a reduction in P_{aCO_2} for a given level of ventilation, but also a reduction in ventilation for any level of P_{aCO_2}. The very low V_T required to keep some patients on the linear portion of the pressure-volume curve and the fact that even such rigorous parameters may not eliminate overdistention injury in some patients[56] highlight this potential utility of TGI. Though there are a great many subtleties to its administration and effect, and valid concerns over potential complications, TGI appears in general to be well tolerated.[115–118]

Beyond Acute Respiratory Distress Syndrome

The preceding discussion has emphasized the hazards of VILI, the importance and demonstrated efficacy of lung-protective ventilatory strategies, and the issues surrounding attendant respiratory acidosis, with particular reference to ARDS. While there are special practical, scientific, and historical relationships between ARDS and these management concerns, the evolving lessons, including the potential benefits of TGI, are certain to have broader applicability, and to obtain to a variety of disease states. Status asthmaticus is an obvious example, and indeed represented one of the first effective applications of volume-limited ventilation and permissive hypercapnia.[65] The approach seems naturally extensible to other causes of respiratory failure, particularly those characterized by inhomogeneity and increased susceptibility to VILI. As the essential components, benefits, and risks of protective strategies become better delineated, they may inform management in chronic obstructive pulmonary disease, necrotizing pneumonia, and lung transplantation.

Tracheal gas insufflation has been suggested for entirely different clinical applications. Its potential to facilitate ventilator liberation by increasing the efficiency of CO_2 clearance and decreasing work of breathing has been frequently mentioned. Though there may be merit to such an approach in specific cases, its routine use should be viewed circumspectly. Though TGI might, for instance, accelerate extubation or decannulation of a chronic obstructive pulmonary disease patient recovering from hypercapnic respiratory failure, its augmentation of efficiency ends at that moment. Such an approach seems akin to the mistake of ventilating the chronic CO_2 retainer to eucapnia. Unless there is a transition to another TGI technique, such as gas administration through a SCOOP catheter (Transtracheal Systems, Englewood, Colorado), independent ventilation might be expected to be short-lived. The valid enthusiasm over the use of TGI in some clinical settings should not degenerate into a case of a technology in search of an application.

Summary

VILI has become recognized as an important factor exacerbating primary lung injury in acute respiratory failure. Lung-protective ventilatory strategies are of proven mortality benefit in ARDS, presumably through reduction of VILI. The attendant hypercapnia and respiratory acidosis are thought to be generally well tolerated, but are contraindicated in certain patient populations; the more sobering possibility that they are in fact harmful in as yet indeterminate ways has recently regained attention. TGI may provide a means by which to deploy protective ventilatory strategies while limiting hypercapnia and acidosis, with numerous advantages over bicarbonate and other therapies.

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Animal and Lung Model Studies of Tracheal Gas Insufflation

Avi Nahum MD PhD

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Tracheal gas insufflation (TGI) is the continuous or phasic insufflation of fresh gas into the central airways for the purpose of improving the efficiency of alveolar ventilation and/or minimizing the ventilatory pressure requirements. Fresh gas is insufflated near the main carina, usually at flow rates of 2–15 L/min. During expiration, TGI clears the anatomic and apparatus dead space proximal to the catheter tip, thus improving carbon dioxide (CO_2) clearance. Moreover, at high catheter flow rates turbulence generated at the tip of the catheter may enhance distal gas mixing. CO_2 elimination during TGI depends on catheter flow rate, as at higher flow rates a greater portion of the proximal dead space is flushed clear of CO_2. Consequently, as TGI flow is increased, arterial carbon dioxide tension (P_{aCO_2}) decreases. Eventually, with increasing catheter flow rate, fresh gas completely flushes the available dead space during expiration and the P_{aCO_2} reaches a plateau. At that point, increasing catheter flow rate decreases P_{aCO_2} much less, probably because of turbulent mixing in the airways distal to the catheter tip. In clinical practice, TGI can be applied either to decrease P_{aCO_2} while maintaining tidal volume constant or to decrease tidal volume while keeping P_{aCO_2} constant. In the former strategy, TGI is used to protect pH, whereas in the latter it is used to minimize the stretch forces acting on the lung parenchyma, to minimize ventilator-associated lung injury.

Key words: tracheal gas insufflation, animal model, lung model, lung mechanics, mechanical ventilation, airway pressures. [Respir Care 2001;46(2):149–157]

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Introduction

Tracheal gas insufflation (TGI) is the continuous or phasic insufflation of fresh gas into the central airways for the purpose of improving the efficiency of alveolar ventilation and/or minimizing the ventilatory pressure requirements. Recently, TGI has received increasing attention as an adjunctive tool to mechanical ventilation. Experimental studies both in lung models and animal models have been essential in understanding TGI's mechanisms of action and operational characteristics. Moreover, experimental studies, especially in animal models of lung injury, have substantiated the potential role of TGI in clinical practice.

Mechanisms of Action

The efficacy of conventional tidal breaths during TGI is improved primarily by two mechanisms.[1–3] First, fresh gas introduced by the catheter during expiration can dilute the carbon dioxide (CO_2) stored in the series (anatomic) dead space compartment proximal to the catheter tip (Fig. 1). Second, at high catheter flow rates, turbulence generated at the tip of the catheter can enhance gas mixing in regions distant to the catheter tip, thereby contributing to CO_2 removal. TGI is unlikely to be very effective when the alveolar (as opposed to the series compartment) dominates the total physiologic dead space (the ratio of the dead space to the tidal volume, V_D/V_T); yet at small V_T (whenever series dead space contributes substantially to V_D/V_T) or when alveolar ventilation is very low, TGI should be helpful. Many investigators have found that combining TGI with conventional mechanical ventilation (CMV) techniques and high-frequency jet ventilation augments CO_2 elimination.[4–10] This approach takes advantage of improved ventilatory efficiency due to anatomic dead space washout as well as enhanced mixing due to catheter turbulence generated to permit reduced V_T.[11] Another promising modality is combining TGI with external chest vibrations. This novel ventilation technique enhances intra-airway mixing (peak CO_2 transport resistance was displaced from second-generation to fourth-generation airways), thus improving CO_2 elimination.[12] In normal dogs, TGI decreased arterial carbon dioxide tension (P_{aCO_2}) from a baseline value of 48 ± 2 mm Hg to 41 ± 2 mm Hg, and addition of high-frequency (29 Hz) chest vibrations further decreased P_{aCO_2} to 33 ± 1 mm Hg.[12]

Operational Characteristics

Modes of Operation

During TGI, delivery of fresh gas occurs either throughout the respiratory cycle (continuous flow) or only during a specific segment of it (phasic flow).[13,14] Continuous catheter flow has also been used in combination with a shutter or a mechanical ventilator, in which closure of an expiratory valve forces catheter flow to deliver all or part of the inspired V_T.[5,7,15] Phasic TGI is delivered selectively during inspiration or expiration. Inspiratory TGI can be used as the only source of fresh gas, thereby bypassing the portion of the anatomic dead space proximal to the catheter tip, or it can be used in combination with a conventional ventilator to augment alveolar ventilation.[13] During expiratory TGI, catheter flow is timed to occur during all or part of expiration, augmenting alveolar ventilation by flushing CO_2 from the central airways and apparatus dead space.

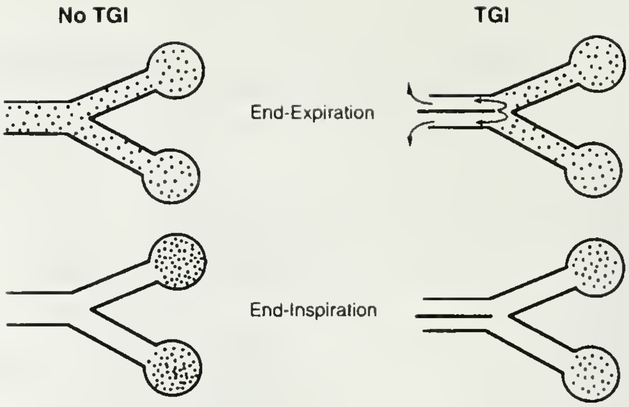

Fig. 1. Primary mechanism of action of tracheal gas insufflation (TGI). At end-expiration, in the absence of TGI (upper left), central airways are laden with carbon dioxide (CO_2) (dots). This exhaled CO_2 is then recycled back into the alveoli during the next inspiration (lower left), thus limiting CO_2 elimination. With TGI, CO_2 is flushed out of the central airways during expiration, so less CO_2 is recycled back to the alveoli, effectively decreasing anatomic dead space and increasing CO_2 elimination. (From Reference 4, with permission.)

Carbon dioxide elimination during TGI depends on catheter flow because fresh gas flushes a greater portion of the proximal dead space at higher flow rates (Fig. 2). The volume of fresh gas introduced into the trachea during TGI depends on expiratory time (T_E) and catheter flow ($\dot{V}_c$). At a certain $T_E \times \dot{V}_c$, fresh gas completely sweeps the proximal anatomic dead space during expiration. At that point, increasing $\dot{V}_c$ further probably does not further dilute the CO_2 residing in the series dead space. This operational characteristic of TGI, and the fact that the P_{aCO_2} decrease caused by reduction in V_D/V_T is much less at lower V_D/V_T, limits the decrement in P_{aCO_2} afforded by TGI at high $\dot{V}_c$.[16] Nevertheless, at high $\dot{V}_c$, P_{aCO_2} continues to decrease with increasing $\dot{V}_c$, but at a slower rate (see Fig. 2).[16] Once the series dead space is flushed completely by the fresh gas during expiration, the flow-dependence of P_{aCO_2} is thought to be secondary to enhanced turbulent mixing in the airways distal to the catheter tip.[1–3]

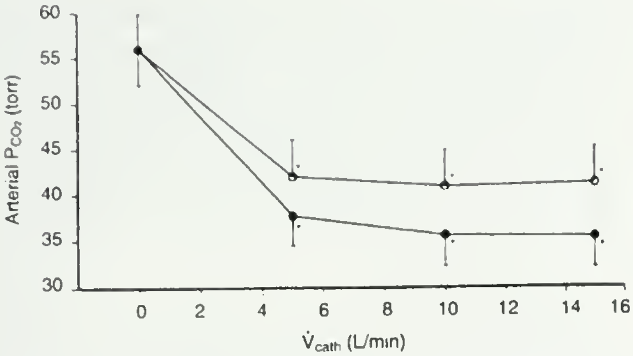

Fig. 2. Effect of tracheal gas insufflation (TGI) on arterial carbon dioxide tension (P_{aCO_2}) in normal dogs. As catheter flow rate ($\dot{V}_{cath}$) increases, P_{aCO_2} decreases. The effect of TGI on P_{aCO_2} diminishes at higher flow rates because most of the anatomic dead space proximal to the catheter tip is flushed free of carbon dioxide (CO_2). The additional P_{aCO_2} decrement observed with the straight catheter (as compared to the inverted catheter) is thought to be due to distal turbulence with enhanced mixing. torr = mm Hg. (From Reference 3, with permission.)

Because the major mechanism of TGI is flushing the proximal anatomic dead space free of CO_2, if lung deflation takes place throughout expiration secondary to dynamic hyperinflation, the CO_2 that is constantly exhaled by the lung can decrease the efficacy of TGI. This possibility was tested in normal dogs, and the data suggest that the volume of fresh gas insufflated by the catheter, rather than the flow, determines the efficacy of TGI.[14,17] Consequently, to maintain CO_2 elimination efficacy during inverse ratio ventilation, progressively higher flow rates may need to be applied as T_E is shortened.

Catheter Location

A greater portion of the anatomic dead space proximal to the catheter tip during exhalation can be flushed by the fresh gas with more distal catheter placement.[3,18] Moving the catheter toward the carina also advances the turbulence zone generated by the catheter closer to the periphery during both phases of the respiratory cycle, thereby improving the efficacy of TGI. In normal dogs the exact location of the catheter tip (within a few centimeters of the carina) did not prove to be crucial to the efficacy of the TGI in augmenting alveolar ventilation.[3] This observation may simplify the clinical application of TGI, because bronchoscopically or radiographically guided catheter positioning in critically ill patients may not be necessary.

Effect of Tracheal Gas Insufflation on Lung Volume

End-expiratory lung volume (functional residual capacity or FRC) is increased during TGI because of three factors.[3,9] First, part of the momentum of the discharging jet stream is transferred to the alveoli.[19] Second, placement of the catheter within the trachea decreases its cross-sectional area, increases expiratory resistance, and delays emptying. Third, catheter flow through the endotracheal tube, expiratory circuit, and expiratory valve during expiration can build a back pressure that impedes expiratory flow from the lung. The first and third mechanisms are very similar to what happens during constant flow ventilation. Brampton and Young[17] showed that most of the alveolar pressure generated during constant flow ventilation in dogs was due to the latter mechanism. At flow rates of 8–60 L/min they estimated that only 1.3 cm H_2O of pressure was generated at the alveolar level secondary to momentum transfer. Consequently, the study suggests that most of the dynamic hyperinflation produced by TGI is secondary to decreased surface area of the trachea and outflow resistance to gas flow.

The rise in FRC during TGI is controlled by adjusting the ventilator-set positive end-expiratory pressure (PEEP) level. In order to maintain FRC constant between TGI and no TGI conditions, ventilator-set PEEP is decreased during TGI such that total PEEP (ie, ventilator-set PEEP plus intrinsic PEEP [auto-PEEP]) remains constant.[3,18,20] Alternatively, the effect of TGI on lung volume can be controlled to a certain extent by using reversed[3,21,22] or reverse-thrust catheters.[15] Reversing the flow so that the jet leaving the catheter tip discharges mouthward creates a Venturi effect that decreases dynamic hyperinflation caused by TGI (Fig. 3). However, in certain situations this effect can be large enough to actually decrease FRC during TGI. In that case, ventilator-set PEEP may need to be increased to counterbalance the effect of the reverse-thrust jet if decreased FRC is undesirable.

Catheter Design

Currently there is not a standard method of introducing the insufflation catheter into the trachea. In most studies a small-caliber catheter was introduced through an angled side-arm adapter attached to the endotracheal tube (ETT) and positioned just above the main carina.[9] Catheter placement was usually performed with bronchoscopic guidance or estimated from a recent chest radiograph. This type of system is simple to construct and can be duplicated in most laboratories, but suffers from some drawbacks in the clinical setting. New ETT designs that incorporate channels within the endotracheal tube wall may solve many of the problems and simplify application of TGI[23,24] and can be used to perform TGI in patients. Future clinical applications of TGI will probably use a modified ETT that incorporates the catheter in its wall, attached to a standardized circuit for gas delivery. The design and application of various TGI circuits are discussed in detail elsewhere in this issue of RESPIRATORY CARE.[25]

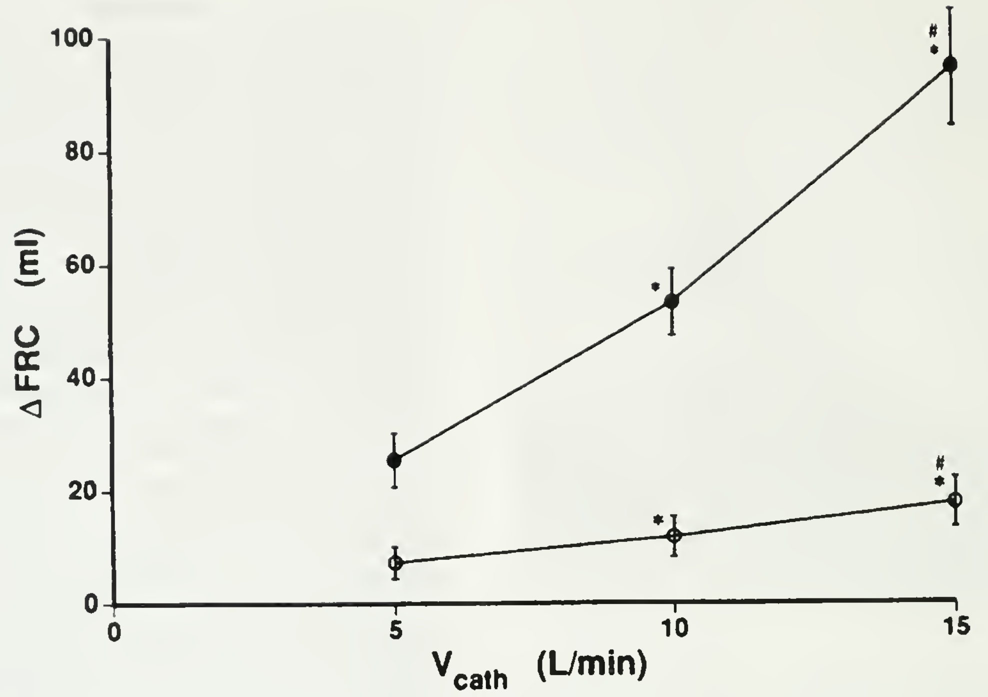

Fig. 3. Tracheal gas insufflation increases end-expiratory lung volume (ie, functional residual capacity or FRC) in a flow-dependent manner in normal dogs. The rise in FRC (ΔFRC) is less with increasing catheter flow rate ($\dot{V}_{cath}$) with a straight catheter than with an inverted catheter. The tips of both catheters were placed at the same location, approximately 2 cm above the main carina (* significantly different from 5L/min [$p < 0.05$]; # significantly different from 10 L/min [$p < 0.05$]). (From Reference 3, with permission.)

Monitoring during Tracheal Gas Insufflation

Airway Opening Pressure and Lung Mechanics

Under baseline conditions, mean airway opening pressure ($\bar{P}_{ao}$) and mean alveolar pressure ($\bar{P}_A$) are related by the following expression:

$$\bar{P}_{ao} = \bar{P}_A + \{\dot{V}_E/60 \times (R_E - R_I)\}$$

where R_E and R_I are the expiratory and inspiratory resistances, respectively, and $\dot{V}_E$ is the minute ventilation.[26] Consequently, during mechanical ventilation, monitoring $\bar{P}_{ao}$ gives useful information regarding $\bar{P}_A$. During TGI, however, the jet stream increases flow through the ventilator circuit during expiration and creates a region of bidirectional flow within the airways. Both effects change the resistance characteristics of the respiratory system and modify the relationship between $\bar{P}_{ao}$ and $\bar{P}_A$ observed at baseline (ie, when $\dot{V}_c$ = 0 L/min). Since R_E increases during TGI, $\bar{P}_{ao}$ tends to underestimate $\bar{P}_A$ (and end-expiratory lung volume) when the system is switched from baseline to TGI conditions. In an experimental study, monitoring tracheal pressure 2 cm beyond the tip of the catheter seemed to accurately gauge lung volume changes at end-expiration, suggesting that tracheal pressures should be monitored beyond the jet stream during TGI.[3] During pan-expiratory TGI, catheter flow ceases during inspiration, and inspiratory P_{ao} provides useful information regarding P_A, as during CMV. In contrast, during continuous TGI, inspiratory $\bar{P}_{ao}$ (measured at the tip of the endotracheal tube) differs from the tracheal pressure (P_{trach}, measured distal to catheter orifice). The magnitude of $P_{ao} - P_{trach}$ during inspiration depends on the inspiratory flow through the circuit and circuit geometry spanning the two pressure measurement points, but is usually less than 3 cm H_2O. As the ventilator-set V_T is decreased with increasing $\dot{V}_c$, the difference between P_{ao} and P_{trach} approaches zero. However, during expiration under both continuous and expiratory TGI conditions, catheter flow pressurizes the respiratory system and, as a result, P_{ao} underestimates P_{trach}. The magnitude of $P_{ao} - P_{trach}$ during expiration increases with $\dot{V}_c$ and probably depends on the geometry of the system and the orientation of the catheter with respect to the trachea.

TGI may also interfere with the clinician's ability to measure lung mechanics. Respiratory system compliance and auto-PEEP measurements require application of a pause at end-inspiration and/or at end-expiration. If catheter flow continues during these measurements, the pressure within the respiratory system builds up over time. Consequently, a plateau pressure cannot be obtained and, if unnoticed, alveolar pressure can increase to hazardous levels. During pan-expiratory TGI, depending on the timing and nature of the signal that gates the solenoid to divert catheter flow to the atmosphere, these measurements can still be made safely.[13,22] Nevertheless, it is advisable to test the TGI-ventilator system with a mechanical lung model under controlled conditions prior to measuring lung mechanics at the bedside.

Tidal Volume

Whenever catheter flow is delivered during inspiration, it contributes to total inspired V_T. The contribution to total inspired V_T is eliminated if TGI is timed to occur only during expiration.[13] Even then decompression of the TGI circuit into the ventilator circuit during the inspiratory phase of solenoid closure contributes to total inspired V_T.[20,27] In most TGI circuits, however, this volume is rather small (10–20 mL at $\dot{V}_c$ of 10 L/min). These problems are avoided if an independent measure of V_T, such as inductive plethysmography, is used. The effect of TGI on total inspired V_T depends on the ventilator operation mode.

Flow-Controlled Volume-Cycled Ventilation

During continuous TGI, total inspired V_T is composed of two components: that delivered by the ventilator (V_{Tv}) and that delivered by the catheter (V_{Tc}). That is, $V_T = V_{Tv} + V_{Tc}$. The contribution of continuous TGI to total inspired V_T can be estimated from the duration of inspiration (inspiratory time or T_I) and $\dot{V}_c$, because $V_{Tc} = T_I \times \dot{V}_c$. Consequently, during flow-controlled volume-cycled ventilation, total inspired V_T can be maintained relatively constant during continuous TGI by decreasing the ventilator-set V_T by an amount equal to V_{Tc}.[20]

Pressure-Control Ventilation

During pressure-control ventilation (PCV), application of TGI does not change the total inspired V_T, provided TGI does not pressurize the respiratory system beyond the set-pressure (P_{set}, defined here as the end-inspiratory pressure during PCV). As $\dot{V}_c$ is increased, the ventilator-delivered V_T declines, but the total inspired V_T remains the same.[8,28] The respiratory system behaves in this manner as long as V_{Tc} is less than the V_T generated by P_{set} under PCV conditions without TGI. If V_{Tc} exceeds the V_T generated by PCV in the absence of TGI, then TGI will overpressurize the circuit and peak P_{ao} will be greater than that produced by the ventilator-set pressure. Most ventilators allow pressures higher than P_{set}, as long as P_{ao} remains below the high pressure limit of the ventilator. Consequently, excessive pressures can be produced within the respiratory system if the product $\dot{V}_c \times T_I$ is too large.[9,22,29]

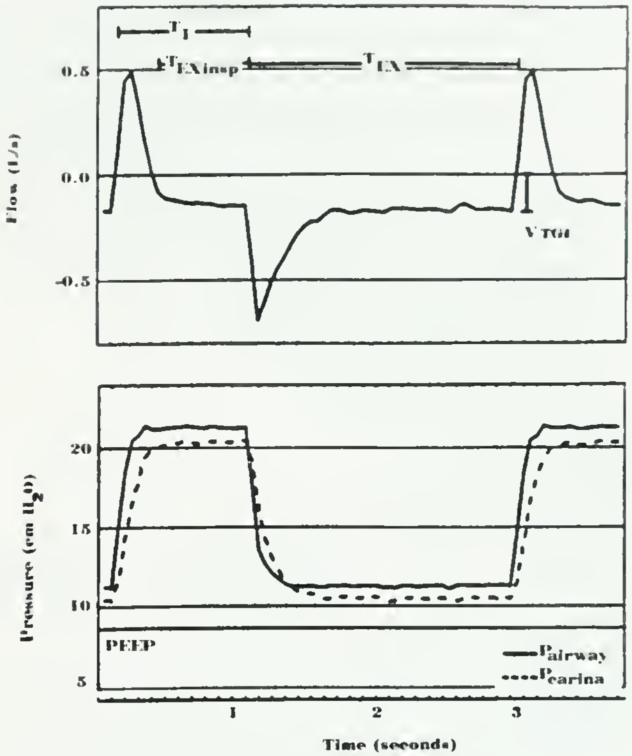

Fig. 4. Flow (top graph) and pressure (bottom graph) tracings from a sheep with lung lavage-induced lung injury, demonstrating the release of tracheal gas insufflation (TGI) flow during inspiration. During inspiration, both airway opening pressure (solid line) and tracheal pressure (dotted line) reach a plateau. However, during the latter part of inspiration (T_{EXinsp}), an expiratory valve allows TGI flow to discharge to the atmosphere (note that flow becomes negative during T_{EXinsp}). Lung deflation starts at the end of inspiration (T_I) when flow through the circuit becomes equal to TGI flow (V_{TGI}). PEEP = positive end-expiratory pressure. (From Reference 22, with permission.)

When this happens, the P_{ao} time profile becomes a hybrid of PCV and constant-flow volume-cycled ventilation, resembling that generated during volume-assured pressure support ventilation. This problem can be circumvented by introducing a pressure-release valve into the ventilator circuit that dumps circuit pressure above a set threshold.[29] New generation ventilators with active valves that allow expiratory flow during inspiration when P_{ao} exceeds P_{set} eliminate this problem by discharging TGI flow to the atmosphere when $P_{ao} > P_{set}$ (Fig. 4).[22]

Monitoring Carbon Dioxide Elimination Efficacy of Tracheal Gas Insufflation

TGI modifies the profile of the expired-gas capnogram because fresh gas delivered by the catheter dilutes the CO_2 exhaled from the lungs. The capnogram measures exhaled CO_2 at the tip of the ETT and therefore monitors the exhaled CO_2 with a time (and volume) lag relative to the catheter tip. Nevertheless, the exhaled-gas capnogram can be used qualitatively to gauge the completeness of expiratory washout. Clearly, if end-tidal carbon dioxide tension (P_{ETCO_2}) declines to very low values (< 3 mm Hg), expiratory washout is probably complete, and further increments in $\dot{V}_c$ may not impact P_{aCO_2} greatly. However, if more CO_2-laden gas is removed from the periphery of the lung by the distal effects of TGI as $\dot{V}_c$ is increased, P_{ETCO_2} may remain elevated. Consequently, the relationship between the efficacy of TGI and the capnographically measured CO_2 profile is quite complex. Nevertheless, certain observations can be made based on capnography.

The fraction of unperfused alveoli can be estimated in terms of P_{aCO_2} and average P_{ETCO_2} as: $(P_{aCO_2} - P_{ETCO_2})/P_{aCO_2}$.[30] This ratio can be readily measured at the bedside by capnography and arterial blood gas analysis. Since increasing the fraction of unperfused alveoli decreases the efficacy of TGI (as measured by percentage change in V_D and P_{aCO_2} relative to baseline conditions), the ratio $(P_{aCO_2} - P_{ETCO_2})/P_{aCO_2}$ may provide useful clinical information. Indeed, in both animal[16] and human[31] studies this ratio was closely correlated ($r = 0.70$) with percentage reduction in P_{aCO_2} from baseline during TGI. The exact role of capnography in monitoring the efficacy of TGI remains to be established, but available data suggest that it may be used to optimize $\dot{V}_c$ and ventilator settings.

Tracheal Gas Insufflation and Ventilator Interactions

Since TGI introduces an external flow source independent of the ventilator, it can adversely affect the ventilator's ability to monitor pressures and volumes and may cause the ventilator to alarm incessantly. The presence of catheter flow during expiration disables the monitoring role of the expiratory pneumotachograph of the ventilator, causing some ventilators to alarm when the difference between the measured inspired volume and the exhaled volume exceeds a certain value. More importantly, the presence of an external flow that can pressurize the ventilator circuit interferes with the ventilator's ability to detect a leak. Incompatibilities between the ventilator and TGI can usually be eliminated in external TGI systems during expiratory TGI if flow and P_{ao} are monitored at the tip of the ETT. Alternatively, internalizing the TGI circuit into the ventilator allows the ventilator to account for the TGI flow and can simplify ventilator-TGI interactions.

Clinical Implications

Carbon Dioxide Elimination

The effect of a given TGI-induced change in V_D and P_{aCO_2} depends on the P_{aCO_2} and V_D/V_T values prior to the initiation of TGI, and on the effect of TGI on CO_2 production.[8] For a given fractional change in V_D, the percentage change in P_{aCO_2} increases dramatically as V_D/V_T exceeds 0.70.[9] This implies that the effect of TGI on P_{aCO_2} is amplified as the respiratory system is allowed to operate at higher V_D/V_T (ie, permissive hypercapnia). Consequently, TGI becomes more effective in decreasing P_{aCO_2} in the setting of hypercapnia.[9]

In the setting of acute lung injury, part of the dead space resides in the alveoli as alveolar dead space. The alveolar gas originating from those ventilated but hypoperfused lung regions is CO_2-poor. Consequently, gas expired from alveolar dead space dilutes CO_2-laden gas residing in the proximal anatomic dead space. Consequently, the impact of washing proximal dead space free of CO_2 on alveolar ventilation diminishes.[13] Adopting a permissive hypercapnia strategy increases the amount of CO_2 that can be removed from the proximal anatomic dead space and counterbalances the decreased CO_2 removal efficacy of TGI caused by increased alveolar dead space (Fig. 5). In most studies, when the initial P_{aCO_2} (without TGI) was maintained at 60–80 mm Hg, TGI at 6–10 L/min was able to decrease P_{aCO_2} by 15–30%.[9]

As an adjunct to a pressure-targeted lung-protective ventilatory strategy, TGI can be used to pursue one of two goals. It can be used to limit the extent of hypercapnia and/or to control the rate of rise of P_{aCO_2} while maintaining V_T and minute ventilation constant. Alternatively, it can be used to limit ventilatory distending forces (ie, allowing a reduction in V_T) while maintaining P_{aCO_2} constant. Using TGI in a canine oleic acid-induced pulmonary edema model, adequate alveolar ventilation was maintained at much smaller V_T and pressures than required without TGI.[20] Compared to CMV, the valved TGI catheter (which functioned as the ventilator in this setting) achieved the same ventilatory task at 35% of the V_T and 70% of elastic end-inspiratory pressure. Using a similar strategy, Kolobow et al demonstrated that they could adequately ventilate sheep that had undergone resection of 88% of their lung tissue, without resorting to excessive $\dot{V}_E$ or airway pressures.[5]

Oxygenation

In normal dogs, TGI tended to reduce venous admixture ($\dot{Q}_V/\dot{Q}_T$) and increase arterial oxygen tension.[3,8,28] In this experimental setting, most of the increase in arterial oxygen tension could be explained by the decrement in P_{aCO_2} caused by TGI. In 6 oleic acid-injured dogs, TGI did not improve arterial oxygen tension or $\dot{Q}_V/\dot{Q}_T$ when V_T and FRC were kept constant.[16] Current data suggest that TGI does not impact oxygenation, provided total inspired V_T and FRC are not augmented during application of TGI.

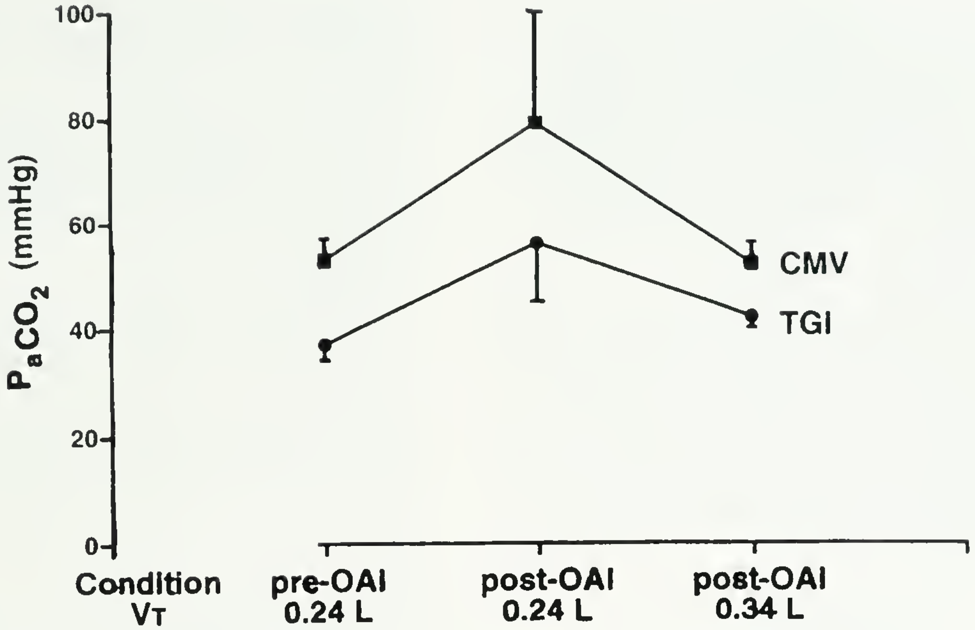

Fig. 5. Effect of tracheal gas insufflation (TGI) on arterial carbon dioxide tension (P_{aCO_2}) before and after oleic acid-induced lung injury (OAI) in dogs during conventional mechanical ventilation (CMV) and TGI. Tidal volume (V_T) during pre-OAI and the first post-OAI stages was kept constant, at 0.24 L. On the other hand, V_T during the second post-OAI stage was increased to 0.34 L to match post-OAI P_{aCO_2} to the values obtained during pre-OAI under CMV conditions. The efficacy of TGI, as judged by the difference in P_{aCO_2} between CMV and TGI conditions, was preserved during OAI when P_{aCO_2} was allowed to rise. Efficacy of TGI deteriorated, however, once P_{aCO_2} was decreased when V_T was increased. (Adapted from Reference 16.)

Work of Breathing

The minute ventilation sparing effect of TGI may also be used to reduce work of breathing in some intubated patients. However, TGI may impair the ability of some patients to trigger the ventilator, because the patient's inspiratory effort must first outstrip catheter flow and overcome the dynamic hyperinflation caused by TGI in order to lower airway opening pressure below the trigger threshold.[32] The net effect of TGI on work of breathing would depend on the interactions between TGI and the ventilator and on the efficiency of TGI in decreasing dead space and $\dot{V}_E$ requirements. Obviously, incorporating TGI into a flow-by system would preserve the combined benefits of flow-triggering and improved gas exchange associated with TGI. Patients who have neuromuscular weakness but relatively normal lung parenchyma and retain CO_2 because they can only generate small V_T (ie, proximal anatomic dead space contributes substantially to V_D/V_T) are excellent candidates for TGI. Reverse-thrust TGI (ie, with catheter flow directed toward the mouth) may also maintain the patency of the ETT by clearing tracheal secretions from the ETT lumen.[33]

Potential Complications

Although a promising adjunct to CMV, TGI is not without potential complications.[4,9] When high flows are delivered into the airways, potentially any obstruction to outflow of gas could cause lung overinflation within seconds and thus cause pneumothorax, pulmonary venous air embolism, and/or hemodynamic compromise. Esophageal pressure and/or chest wall monitoring may be required to monitor changes in lung volume. A second concern is bronchial mucosal damage by impact of the jet stream onto the bronchial mucosa, as well as the possible physical impact of the catheter tip from oscillation resulting from high flow. The force created by the jet stream impacting on the surface can be quite high and accounts for the bronchial mucosal damage observed in experiments during constant-flow ventilation.[33,34] Proper humidification of the inspired gas is essential at such high flow rates. Long-term use of TGI may result in inspissation or retention of secretions, especially if the insufflated gas is not adequately humidified.

Summary and Future Directions

Experimental work over the last decade, both with in vitro and in vivo systems, has tremendously increased our understanding of the usefulness and limitations of TGI as an adjunct to mechanical ventilation. Clearly, the published clinical experience in mechanically ventilated patients is very limited but expanding rapidly. However, none of the clinical studies to date have explored the role of TGI in a lung-protective ventilatory strategy. The logical application of TGI in an acute respiratory distress syndrome patient is to limit P_{aCO_2}, V_T, or both. We are now in a position to safely apply TGI at the bedside to further improve ventilatory management of critically ill patients. However, routine bedside application of TGI awaits carefully performed clinical studies to determine its efficacy and safety profile.

Further experimental work is needed to enhance the operational characteristics of TGI and to develop its full potential in the clinical setting. Certain applications of TGI are yet to be developed, such as drug delivery systems based on TGI and TGI as a weaning tool. Innovations in combined ventilatory modalities, such as TGI with partial liquid ventilation,[35] and utility of TGI in disease processes other than lung injury, such as bronchoconstriction,[34] will also require further testing and development in experimental models.

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Clinical Studies of Tracheal Gas Insufflation

Lluis L Blanch MD PhD

Introduction
Physiologic Effects of Tracheal Gas Insufflation
Efficiency of Carbon Dioxide Removal
Tracheal Gas Insufflation in Patients with End-Stage Pulmonary Disease
Tracheal Gas Insufflation during Mechanical Ventilation: Clinical Studies
Weaning
Monitoring during Tracheal Gas Insufflation
Potential Complications
Summary

Tracheal gas insufflation (TGI) is an adjunct to mechanical ventilation that allows ventilation with small tidal volumes while carbon dioxide (CO_2) is satisfactorily cleared. Pioneering studies in healthy animals and in humans suffering respiratory failure showed that the expiratory flushing of proximal dead space decreased minute ventilation with no change in arterial partial pressure of carbon dioxide (P_{aCO_2}). Recent work indicates that conventional mechanical ventilation aided by TGI may represent a novel ventilatory strategy that succeeds in limiting both the distending forces acting on the lungs and the P_{aCO_2} elevation of permissive hypercapnia. Furthermore, some studies suggest that weaning aided by TGI may allow a reduction in minute ventilation, P_{aCO_2}, physiologic dead space, and the patient's respiratory demands. Clinical use of TGI requires careful monitoring of delivered volumes and pressures to ensure safe clinical application and to evaluate the effect on lung function. Finally, routine use of TGI in intensive care warrants further investigation to solve some technical problems and randonized clinical trials to confirm the beneficial effects in the absence of complications.

Key words: tracheal gas insufflation, clinical studies, mechanical ventilation, ventilator weaning. [Respir Care 2001;46(2):158–166]

Introduction

Extensive animal research suggests that lung injury and inflammatory response may be caused and perpetuated if mechanical ventilation results in alveolar overdistention and allows cyclic collapse and reinflation of alveolar units with tidal breathing. Based on that work, lung-protective mechanical ventilatory strategies have been proposed for acute respiratory distress syndrome (ARDS).[1,2] These strategies typically involve the use of small tidal volume (V_T) to avoid high alveolar pressures at end-inspiration and the resulting alveolar overdistention, and the use of high positive end-expiratory pressure (PEEP) levels to keep alveoli open at end-expiration, thus maintaining alveolar recruitment. Such ventilatory strategies may involve a decrease in alveolar ventilation and a significant increase in arterial partial pressure of carbon dioxide (P_{aCO_2}), a strategy that has been called permissive hypercapnia. Recently, 3 clinical trials showed that treatment with a ventilation approach designed to protect the lungs from excessive stretch resulted in improvements in several important clinical outcomes in patients with acute lung injury and in ARDS patients.[3–5] Unfortunately, carbon dioxide (CO_2) retention must sometimes occur over brief inter-

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vals, which leads to unacceptably severe respiratory acidosis and is contraindicated in some patients.

Although permissive hypercapnia is generally well tolerated, it may not be acceptable in some patients in whom lung injury and brain injury coexist. Based on recent data,[4] it would be advantageous to use a lung-protective strategy without respiratory acidosis. Tracheal gas insufflation (TGI) is an adjunct to mechanical ventilation that allows ventilation with small V_T while CO_2 is satisfactorily cleared.[6] Pioneering studies with healthy experimental animals[7] and with humans suffering respiratory failure[8] found that the expiratory flushing of proximal dead space decreased minute ventilation with no change in P_{aCO_2}. Recent work indicates that conventional mechanical ventilation aided by TGI may represent a novel ventilatory strategy that succeeds in limiting both the distending forces acting on the lung and the P_{aCO_2} elevation of permissive hypercapnia.[9–12]

Physiologic Effects of Tracheal Gas Insufflation

Tracheal gas insufflation applied together with conventional mechanical ventilation effectively reduces the size of the dead space compartment and improves overall CO_2 elimination by flushing with fresh gas the anatomic dead space normally laden with CO_2 during expiration. As a consequence, less CO_2 is recycled to the alveoli during the next inspiration and the ventilatory efficiency of each tidal respiration is improved. Therefore, TGI reduces anatomic dead space and increases alveolar ventilation for a given frequency and V_T combination.[6,13–17]

The main effect of TGI is to flush the dead space from the carina to the Y-piece of the ventilator circuit. TGI also has a distal effect that contributes to CO_2 removal. However, although the distal effect enhances CO_2 removal, the presence of the catheter and the jet effect oppose expiratory flow, favoring intrinsic positive end-expiratory pressure (auto-PEEP).[5,14,18]

Efficiency of Carbon Dioxide Removal

Experimental studies with healthy animals have shown that TGI allows reduction of P_{aCO_2} during hypoventilation.[7,19–21] This effect occurred similarly when the hypoventilation was caused either by a decrease in minute ventilation or an increase in respiratory rate at constant minute ventilation. Application of TGI allowed a decrease in V_T and a 25% reduction in airway pressures. In an experimental model of ARDS, Nahum et al[22] found that increasing CO_2 elimination with 10 L/min of TGI allowed reduction of P_{aCO_2} from 55 mm Hg to 45 mm Hg while V_T was kept constant. However, the efficacy of TGI on P_{aCO_2} diminishes when an increased alveolar component dominates the total physiologic dead space. Nahum et al[23] found that allowing P_{aCO_2} to rise to supranormal levels (a permissive hypercapnia strategy) counteracted the detrimental effect of increased alveolar dead space on the CO_2 removal efficacy of TGI.

CO_2 elimination efficiency is also determined by the position of the tracheal catheter, flush volume, and timing of the tracheal gas flow (continuous or expiratory). Nahum et al[20] found that CO_2 elimination efficiency was greater when the distal tip of the TGI catheter was positioned 1 cm above the carina than when the catheter was placed more proximally.

The volume of gas injected per breath during expiration determines TGI efficiency. In a series of experiments, Ravenscraft et al[9] found that, with or without lung deflation, the volume of gas flushed during the expiratory period determined the effectiveness of TGI, provided that inspired minute ventilation remained unchanged and end-expiration was included in the catheter flush period. Increasing catheter flow in clinical situations where only a brief expiratory time is available may maintain TGI efficiency. In fact, Nakos et al[24] observed an inverse correlation between respiratory rate and P_{aCO_2}, which indicates that longer expiratory times (or lower breathing frequencies) favor TGI efficiency (defined as reductions in P_{aCO_2} and the ratio of dead space to tidal volume $[V_D/V_T]$). Moreover, for the same catheter injection flow and duration, the efficiency of TGI is much higher when catheter flushing is performed late in expiration.[13] That investigation indicated that TGI efficiency is improved by including the end-expiratory segment of the cycle. If TGI is applied only early during expiration, the injected fresh gas is swept out with the discharging alveolar gas. TGI during late expiration flushes CO_2 from the anatomical dead space that would normally remain to be rebreathed on the next inspiration (Fig. 1).

Tracheal Gas Insufflation in Patients with End-Stage Pulmonary Disease

Continuous insufflation of fresh gas (oxygen/air) through an intratracheal catheter has been used in patients with end-stage pulmonary disease to provide continuous oxygen therapy and to decrease oxygen flow requirements.[25–28] In patients with end-stage lung disease, TGI provides a method for oxygen delivery and confers the additional benefits of decreasing dyspnea and increasing exercise tolerance.[29] The physiologic basis for these changes appears to relate to alterations in breathing pattern and gas exchange efficiency.

In tracheostomized, spontaneously breathing patients, Bergofsky and Hurewitz[25] studied the physiologic effects of airway insufflation in patients with chronic CO_2 retention. A continuous flow of 4–5 L/min delivered to the tracheostomy tube produced a reduction in dead space, V_T, and minute ventilation without affecting P_{aCO_2} in the acute state; in the chronic state it maintained or reduced P_{aCO_2}, presumably be-

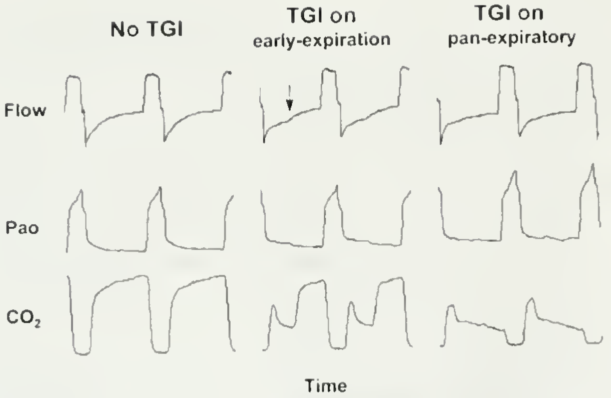

Fig. 1. Flow, airway pressure (Pao), and exhaled capnograms (CO_2) in a mechanically ventilated patient with acute lung injury. Tracheal gas insufflation (TGI) applied early in expiration cannot completely flush CO_2 from the anatomical dead space. CO_2 efficiency is improved when TGI is applied including the end-expiratory segment of the cycle.

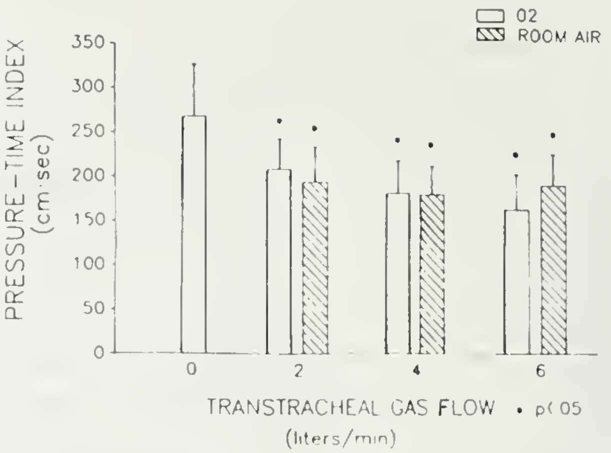

Fig. 2. Transtracheal delivery of oxygen or air reduced the pressure-time index of the diaphragm in 5 patients with end-stage pulmonary disease. (From Reference 27, with permission.)

cause of reduced dead space. Those authors also found reduced oxygen consumption and CO_2 production with airway insufflation in patients who had the most severe form of chronic obstructive pulmonary disease. The decrease in metabolic rate without a change in P_{aCO_2} suggested a reduction in the work of breathing (WOB) during TGI in spontaneously breathing patients. Interestingly, Hurewitz et al[26] found that the application of airway insufflation in patients with advanced obstructive and restrictive lung disease produced either a reduction in minute ventilation with minimal changes of alveolar ventilation, or no variations in minute ventilation associated with an increase in alveolar ventilation. These different responses to dead space reduction suggest that breathing pattern is CO_2-driven in a subgroup of chronic patients, whereas other patients exhibit a breathing pattern modulated by factors such as lung parenchymal receptors, diaphragmatic tension, and/or hyperinflation.[30,31]

In addition to the effects on gas exchange, minute ventilation, and dead space, TGI resulted in decreased oxygen cost of breathing in spontaneously breathing patients. Benditt et al[27] found that transtracheally administered gas reduced the oxygen cost of breathing (estimated by the pleural pressure time index) and changed the respiratory pattern of the diaphragm (diaphragm tension time index) to a less demanding pattern (Fig. 2). These findings helped to explain the improvements in exercise tolerance and decreased dyspnea and supported the use of transtracheal gas therapy for indications other than oxygenation.

Tracheal Gas Insufflation during Mechanical Ventilation: Clinical Studies

A number of studies have been published on the effect of TGI in patients receiving mechanical ventilation. The majority of the studies have been performed in ARDS patients and focused to demonstrate a reduction in V_T and consequently on airway pressure while P_{aCO_2} was maintained constant or reduced during permissive hypercapnia.[9–12,32] Despite the fact that TGI in patients receiving mechanical ventilation was first tested in 1969,[8] level 1 studies are still lacking.

In 1969, Stresemann et al[8] tested the efficacy of continuous and expiratory TGI in two patients with inspiratory positive pressure breathing and observed reduced P_{aCO_2} at 18 L/min of insufflated tracheal flow. This was the first evidence that washout of anatomical dead space was useful in acute respiratory failure. In patients with a variety of lung disorders, Ravenscraft et al[9] studied the effect of continuous flows (2, 4, or 6 L/min) delivered through a catheter positioned 1 cm or 10 cm above the carina. Over the range of flows tested, CO_2 production and peak and mean airway pressures did not change, and P_{aCO_2} exhibited a 15% reduction with the best combination of insufflated flow (6 L/min) and distance (1 cm from the carina) (Fig. 3). A similar study performed by Saura et al[11] also found that a continuous inspiratory flow of 6 L/min permitted a 20% reduction of dead space and P_{aCO_2}. Remarkably, at 6 L/min TGI flow, plateau pressures slightly increased, suggesting that a certain degree of hyperinflation was caused by TGI.

In ARDS patients, part of the dead space resides in the alveoli as alveolar dead space. The alveolar gas originating from those ventilated but hypoperfused lung regions is CO_2-poor, diminishing the impact on alveolar ventilation of washing proximal dead space free of CO_2. Adopting a permissive hypercapnia strategy increases the amount of CO_2 that can be removed from the proximal anatomic dead space and counterbalances the decreased CO_2 removal efficacy of TGI caused by increased alveolar dead

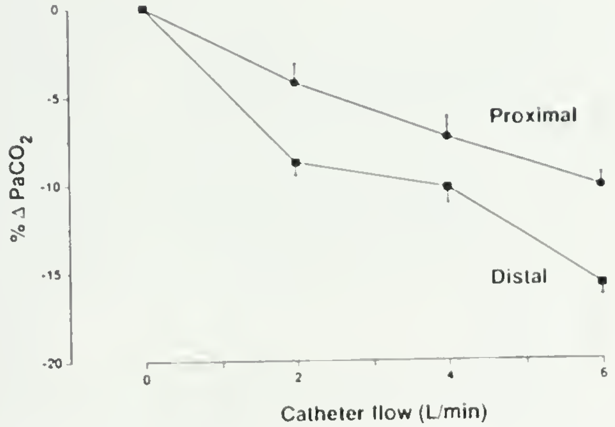

Fig. 3. Percent reduction in arterial partial pressure of carbon dioxide (P_{aCO_2}) from the baseline value as a function of catheter flow. Distal and proximal catheter positions were 1 cm and 10 cm above the carina, respectively. Increasing catheter flow from 2 L/min to 6 L/min caused a reduction in the percent change of P_{aCO_2} from baseline at both catheter positions. (From Reference 9, with permission.)

space.[6] Several studies[10,12,33] have shown that one of the most important features of TGI is to maintain normocapnia or a given level of P_{aCO_2} while V_T is decreased, allowing a reduction in minute ventilation. TGI can thereby be used to decrease the forces acting on the lung, thus minimizing ventilator-induced lung injury in ARDS patients (Fig. 4).

During permissive hypercapnia in ARDS patients, Kalfon et al[32] found that expiratory washout of 15 L/min was extremely useful to reduce P_{aCO_2} (by 30%) and to significantly increase arterial partial pressure of oxygen (from 205 mm Hg to 296 mm Hg). The reduced P_{aCO_2} accompanied an increase in plateau pressure, from 26 cm H_2O to 32 cm H_2O. Substantial increase in airway pressure and lung volume is a well-known side effect of TGI and correlates with the flow used.[20,34] When insufflation of gas is limited to the expiratory phase, V_T remains virtually unchanged during volume-control ventilation, but airway pressures can still increase through expiratory flow limitation and auto-PEEP. Increase in lung volume caused by TGI application is a serious limitation of the technique and should be avoided. Solutions to minimize expiratory TGI-induced auto-PEEP include using lower TGI flows, delivering TGI during pressure-control ventilation, and optimizing mechanical ventilation during TGI application. During pressure-control ventilation, TGI-induced increase in airway pressure automatically results in decreased V_T, and the lack of expiratory TGI-induced auto-PEEP is associated with reduced efficiency of CO_2 elimination.[35] Likewise, if TGI flow is reduced, the ability to clear CO_2 is also diminished.

Optimization of mechanical ventilation appears to be a suitable method to deliver a pressure-limited ventilatory strategy combined with TGI while avoiding auto-PEEP and the deleterious effects of hypercapnia in patients with

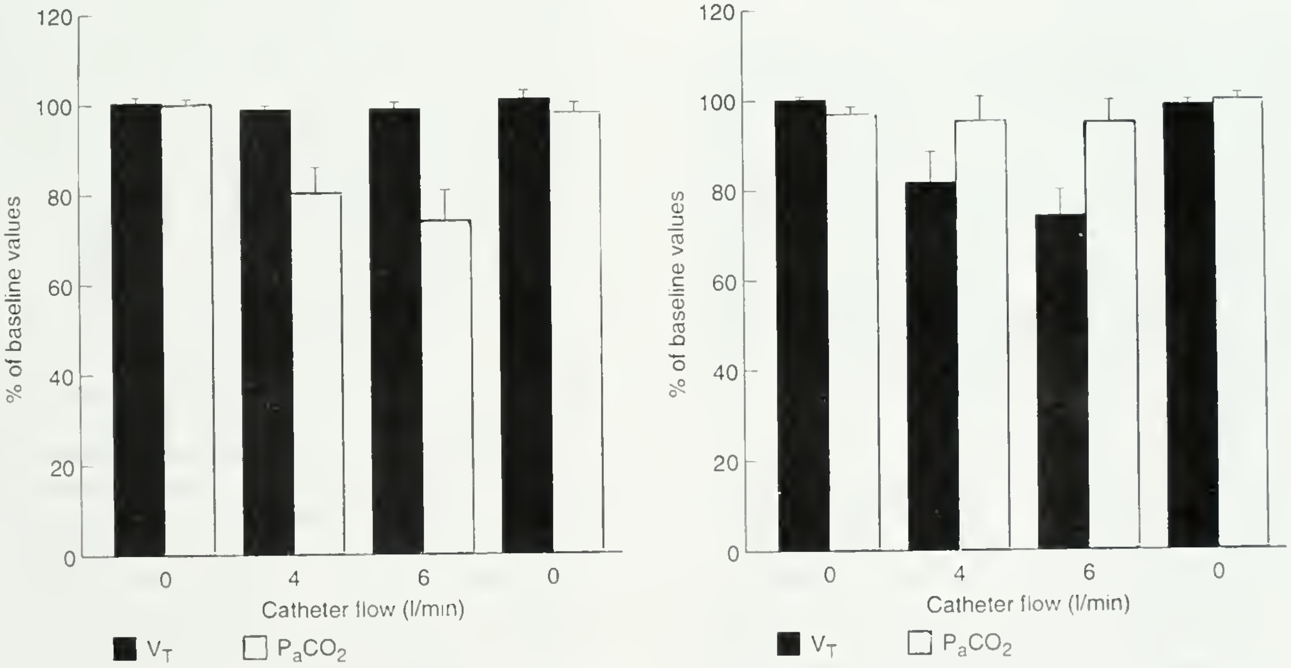

Fig. 4. Left panel: Effect of catheter flow on arterial partial pressure of carbon dioxide (P_{aCO_2}) while tidal volume (V_T) was maintained constant. Right panel: Percent reduction in V_T from baseline value as a function of catheter flow, while P_{aCO_2} was maintained constant. Tracheal gas insufflation can be used to decrease P_{aCO_2} or to minimize the forces (alveolar pressure) acting on the lungs. (From Reference 10, with permission.)

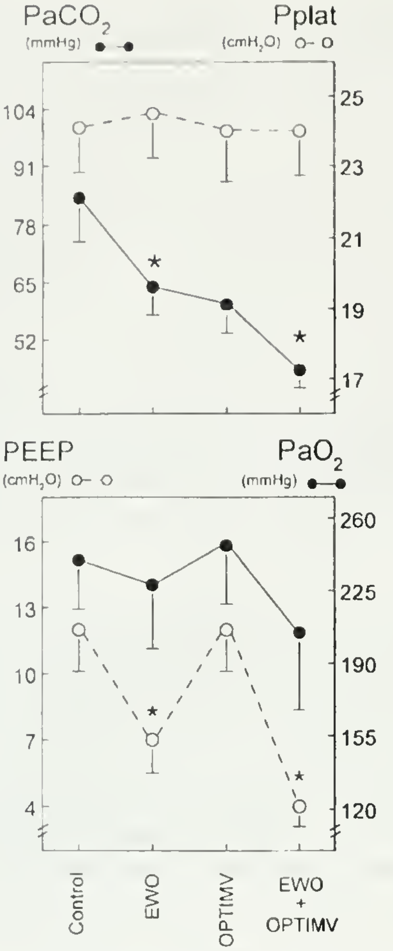

Fig. 5. Changes in arterial partial pressure of carbon dioxide (P_{aCO_2}), inspiratory plateau airway pressure (P_{plat}), positive end-expiratory pressure (PEEP), and arterial partial pressure of oxygen (P_{aO_2}) induced by optimized mechanical ventilation (OPTIMV), expiratory washout (EWO), and the combination of OPTIMV and EWO in 6 patients with severe acute respiratory distress syndrome (ARDS). Extrinsic PEEP had to be reduced by 5.3 ± 2.1 cm H_2O during EWO and by 7.3 ± 1.3 cm H_2O during the combination of OPTIMV and EWO, whereas it remained unchanged during OPTIMV alone. P_{plat} did not significantly change, suggesting that lung hyperinflation was not produced. In patients with severe ARDS, the combination of OPTIMV and EWO had additive effects and resulted in P_{aCO_2} levels close to normal values. (From Reference 12, with permission.)

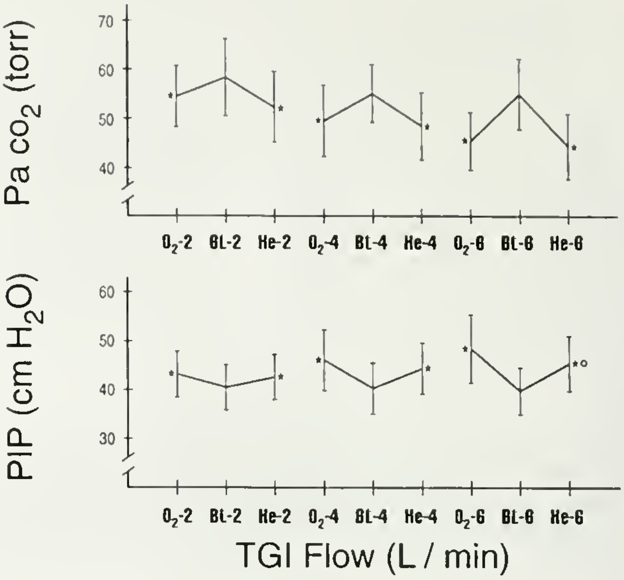

Fig. 6. Arterial partial pressure of carbon dioxide (P_{aCO_2}) and peak inspiratory pressure (PIP) during tracheal gas insufflation (TGI) with oxygen (O_2) and helium (He) at 2, 4, and 6 L/min. BL-2, BL-4, and BL-6 indicate baseline values (no TGI) for comparison purposes. Continuous TGI improved ventilation. At a high flow rate, TGI with helium was associated with a lesser increase in airway pressure than TGI with oxygen. (From Reference 37, with permission.)

severe ARDS. Richecoeur et al[12] found that the combination of increasing respiratory rate to the limit of auto-PEEP, removing the tubing connecting the Y-piece to the endotracheal tube (ETT), and expiratory washout of 15 L/min produced a significant reduction in P_{aCO_2}, from 84 mm Hg during conventional ventilation to 45 mm Hg. The combination of expiratory washout and optimization of mechanical ventilation invariably originated auto-PEEP, but the concomitant decrease in external PEEP allowed a constant total PEEP and plateau pressures during the different treatments (Fig. 5). In a study by Richecoeur et al,[12] oxygenation did not improve, suggesting that expiratory TGI per se has no direct effect on alveolar recruitment when auto-PEEP does not induce an increase in mean airway pressure. The combination of TGI with limited pressure-control ventilation has been proposed as an alternative to extracorporeal or intracaval support for ARDS patients who are highly hypercapnic and hypoxemic.[36]

The effect of different gas mixtures (helium and oxygen) during TGI application has been studied in patients suffering respiratory failure.[37] Helium is an inert gas that has a much lower density than oxygen or air. When given at the same flow as a nitrogen/oxygen mixture, a helium/oxygen mixture produces a much lower Reynolds number and laminar flow.[38] In a study by Pizov et al,[37] TGI with helium was more effective than TGI with oxygen in treating hypercarbia, because the use of helium leads to a lesser increase in airway pressure accompanying the decrease in P_{aCO_2} (Fig. 6). Since the helium/oxygen combination for TGI application has the potential to decrease arterial partial pressure of oxygen, precautions should be taken in the use of helium, particularly in patients who require a high fraction of inspired oxygen to maintain adequate oxygenation.

One of the indications for the use of TGI is the presence of high airway pressures that cannot be reduced as a result of the contraindication of hypercarbia. In the context of lung injury in association with head injury, the aim of TGI is to decrease airway pressures while avoiding the result-

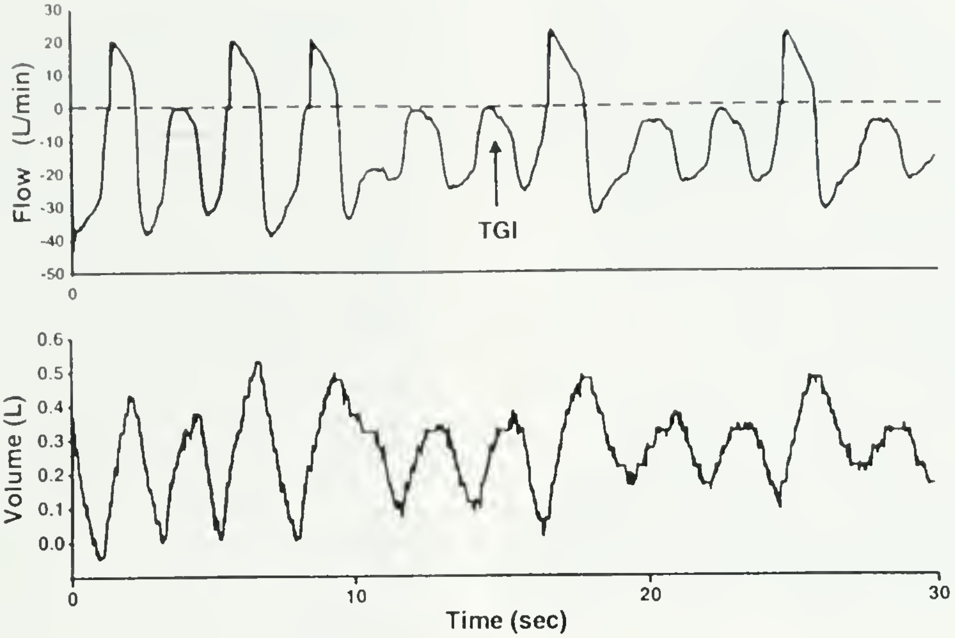

Fig. 7. Flow and inductive plethysmographic lung volume tracing in a mechanically ventilated patient (pressure-support ventilation [PSV] of 4 cm H_2O) during panexpiratory tracheal gas insufflation (TGI) of 8 L/min. In 6 out of 11 breaths, TGI flow met the patient's inspiratory flow demand and provided the total inspiratory volume for the breath and the ventilator was not triggered. (From Reference 46, with permission.)

ing increase in P_{aCO_2}. Elevated intracranial pressure appears to be the only absolute contraindication to permissive hypercapnia.[39] Nevertheless, neurologic patients with head trauma and severe ARDS complicated by barotrauma need a concomitant management of raised intracranial pressure and raised airway pressure. Levy et al[40] found in two patients suffering severe ARDS that the introduction of TGI decreased P_{aCO_2} by 17%, intracranial pressure by 26%, and increased calculated cerebral perfusion pressure. Although the report of Levy et al[40] is anecdotal, it constitutes a firm basis for future research in that type of patient.

Weaning

Liberation from mechanical ventilation is an easy process for most patients. In recent clinical trials, between 60% and 80% of patients could be extubated directly from full ventilatory support.[41–43] However, in patients who have undergone prolonged ventilatory support, weaning can be difficult. Failure of the respiratory muscle pump is probably the most common cause of failure to wean from mechanical ventilation. Indeed, chronic obstructive pulmonary disease patients who subsequently fail a trial of weaning exhibit not only an almost immediate rapid and shallow breathing when ventilatory support is discontinued but also a progressive worsening of pulmonary mechanics, with inefficient CO_2 clearance, in comparison with those chronic obstructive pulmonary disease patients who are successfully extubated after the spontaneous breathing trial. Deterioration of respiratory system mechanics in patients who fail the weaning trial is characterized by increased auto-PEEP, increased inspiratory resistance, and decreased dynamic lung compliance.[44] Thus, inefficient CO_2 clearance in the failing group appeared to be a consequence of worsened pulmonary mechanics with increased energy expenditure and rapid shallow breathing, because the decreased V_T caused an increase in dead space ventilation. In this context, TGI could have a role as an adjunct to ventilator weaning.

Cereda et al[45] studied the effect of TGI combined with continuous-flow positive airway pressure (CPAP) on the effort of breathing in spontaneously breathing sheep with acute lung injury. They found that the beneficial physiologic effects of TGI on minute ventilation and gas exchange were followed by a decrease in the inspiratory WOB. Remarkably, no additional benefit was observed from raising insufflation flow from 10 L/min to 15 L/min. These authors attributed the beneficial effect (of TGI with CPAP) on effort of breathing to a favorable balance between decreased ventilatory requirement and low work load superimposed by the apparatus and TGI. In fact, with CPAP delivered via mechanical ventilator in combination with TGI, additional inspiratory effort is required to overcome the insufflation flow and trigger the ventilator valves. Hoyt et al[46] nicely showed in a bench study that TGI might interfere with ventilator triggering at low peak inspiratory

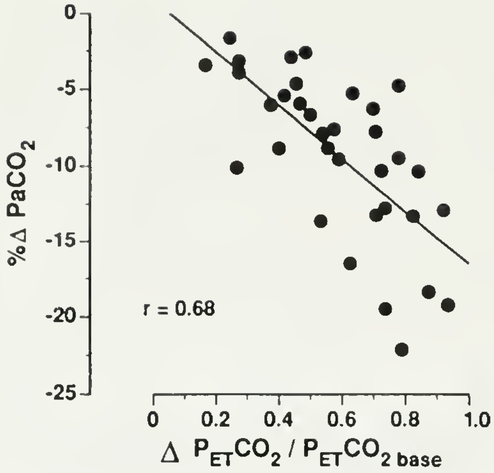

Fig. 8. Percentage reduction in arterial partial pressure of carbon dioxide (P_{aCO_2}) from baseline as a function of the reduction in partial pressure of end-tidal carbon dioxide (P_{ETCO_2}) from the baseline value ($P_{ETCO_{2base}}$). As the difference between P_{ETCO_2} and $P_{ETCO_{2base}}$ widened, larger reductions in P_{aCO_2} were observed. (From Reference 9, with permission.)

flow rates, which suggests that weak patients may fail to open the demand valve of the CPAP system during TGI at high catheter flow rates (Fig. 7).

Based on the hypothesis that TGI has the potential for decreasing anatomical dead space, enhancing gas mixing, and possibly reducing WOB, several authors have tested the effects of TGI on lung function in patients undergoing weaning from mechanical ventilation. A study by Nakos et al[24] found that V_T, minute ventilation, P_{aCO_2}, and physiologic dead space were reduced in a flow-dependent manner when gas was delivered through an oral-tracheal tube. Moreover, they found that distal positioning of the TGI catheter was more effective than proximal positioning and that the effects were less effective in patients with tracheostomy. Interestingly, the improvement in ventilatory efficiency resulting from the functional reduction of dead space allowed for a decrease in P_{aCO_2} at the same respiratory rate and at lower V_T. These data suggest that inspiratory WOB was also reduced, since V_T was significantly reduced. In a preliminary work,[47] we studied the effects of expiratory TGI during CPAP on 6 ready-to-wean patients. Expiratory TGI of 8 L/min decreased the pressure time product of the diaphragm in 5 out of 6 patients, reduced V_T, and caused no changes in respiratory rate or P_{aCO_2}. Data from those studies[24,47] suggest that expiratory TGI might help patients who have difficulties during weaning.

Monitoring during Tracheal Gas Insufflation

TGI is a simple and apparently safe method to reduce both minute ventilation and P_{aCO_2}. Regardless of the approach used, TGI has the potential to alter volumes and airway and alveolar pressure and requires careful monitoring of delivered volumes and pressures to ensure safe clinical application and to evaluate the effect on lung function. Moreover, the position of the TGI catheter inside the ETT should be carefully controlled. The presence of a catheter inside the ETT may increase both inspiratory and expiratory resistance, particularly when small endotracheal or tracheostomy tubes are used.[6,13,14,19] Lucangelo et al[48] studied the air flow resistance of a 2.7 mm external-diameter catheter placed inside ETTs of various sizes and found that resistance differed markedly in the various tube sizes and flows tested.

During continuous TGI, total V_T is the sum of ventilator-derived volume plus the additional volume delivered by TGI. The difference between the pre-programmed ventilator V_T and the ventilator-delivered volume plus the contribution of volume from the TGI catheter can adversely affect the ability of the ventilator to monitor pressures and volumes, calibrations, and leak detections, and may cause the ventilator to alarm non-stop. The measurement of auto-PEEP by the end-expiratory occlusion technique may cause a dramatic elevation of lung volume if TGI flow is not simultaneously interrupted. The same effect would occur with continuous TGI during end-inspiratory occlusion.[6,19]

The efficacy of TGI can be monitored by capnography. Exhaled-gas capnograms provide an indicator of the effect of TGI on the CO_2 concentration of the gas remaining in the proximal anatomic dead space compartment at the onset of inspiration (see Fig. 1).[9,11] Although in patients with respiratory failure the partial pressure of end-tidal carbon dioxide (P_{ETCO_2}) is a poor estimate of P_{aCO_2},[49,50] TGI-induced changes in P_{ETCO_2} correlated significantly with changes in P_{aCO_2}, justifying routine measurement of P_{ETCO_2} during TGI application, as a marker of its effectiveness (Fig. 8).[9,11,23]

Potential Complications

Delivering catheter gas at higher flows needs to be examined with regard to the need for humidification and the potential for tracheal damage with long-term use. The presence of a catheter inside the ETT may complicate suction of respiratory secretions and sputum removal.

TGI has the potential to increase end-expiratory lung volume in a flow-dependent fashion or catheter design.[15,20,21,32,51] Catheter flow can increase alveolar pressure in 3 ways.[6] First, part of the momentum of the discharging jet stream is transferred to the alveoli. Second, placement of the catheter decreases the cross sectional area of the trachea available for expiratory flow, effectively increasing expiratory resistance. Third, catheter flow through the ETT, expiratory circuit, and expiratory valve during expiration builds a back pressure at the airway opening, which impedes expiratory flow from the lung.

Finally, a complete obstruction of the outflow can cause overinflation of the lungs in seconds, with the potential of pneumothorax or hemodynamic compromise.

The development of lung hyperinflation can be effectively controlled by decreasing external PEEP to counterbalance the increase in auto-PEEP so that total PEEP is kept constant.[12] Overpressurization can be identified by examining the airway pressure tracing[13] and can be remedied by placing a pressure-relief valve in the ventilator circuit to dissipate insufflated flow that produces excess pressure.[52] Other authors have designed systems that allow increase of CO_2 clearance without the risks of hyperinflation. De Robertis et al[53] studied the combination of aspiration of anatomic and instrumental dead space in the late part of expiration and replaced with fresh gas through the inspiratory line of the ventilator. This aspiration system allowed reductions in airway pressure and V_T while keeping P_{aCO_2} constant in healthy humans[54] and in ARDS patients.[55] The aforementioned aspiration system has the potential to avoid the problems associated with jet streams of gas or with gas humidification, without developing auto-PEEP. Finally, Takahashi et al[56] observed that the combination of TGI and tracheal gas exsufflation allowed precise control of end-expiratory lung volume while providing the most effective CO_2 elimination.

Summary

Tracheal gas insufflation is a promising complementary technique to mechanical ventilation. TGI is very effective during permissive hypercapnia in ARDS patients, diminishing the complications associated with both mechanical ventilation and respiratory acidosis. Furthermore, some studies suggest that weaning aided by TGI may reduce respiratory demand. However, routine use of TGI in intensive care warrants further investigation to solve some technical problems and to confirm the beneficial effects in the absence of complications with randomized clinical trials in larger groups of patients receiving mechanical ventilation.

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Complications of Tracheal Gas Insufflation

Robert M Kacmarek PhD RRT FAARC

Introduction
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Numerous reports of patient, lung model, and animal use of tracheal gas insufflation (TGI) have appeared in the literature over the past 10 years. However, no commercial TGI system is available. As a result, extreme care must be exercised if attempts are made to provide TGI. Numerous problems with noncommercial systems have been identified. Continuous-flow TGI results in an increase in peak pressure and delivered tidal volume. The use of a flow-limiting or pressure relief valve or a ventilator with an exhalation valve active during exhalation minimizes these problems. Ideally, TGI should only be activated during the expiratory phase. However, this requires that the TGI system be integrated with the mechanical ventilator. In addition, appropriate system monitoring should be available, including measurement of total positive end-expiratory pressure, peak inspiratory pressure, and tidal volume, and there should be a method of identifying increased carinal pressure and deactivating the TGI system if an obstruction occurs proximal to the point of TGI injection. As a result of the potential complications of TGI, this technique cannot be recommended for routine use until commercial systems are available.

Key words: tracheal gas insufflation, complications, mechanical ventilation, positive end-expiratory pressure, PEEP, airway trauma. [Respir Care 2001;46(2):167–176]

Introduction

Over the last 10 years much interest has focused on the potential for mechanical ventilation to induce lung injury similar in pathophysiology to that of acute respiratory distress syndrome.[1] Based on the results of recent clinical trials, it has become the standard of care to ventilate patients with small tidal volumes (V_T) (4–8 mL/Kg) and low peak alveolar pressures (≤ 30–35 cm H_2O).[2,3] However, even if respiratory rate is increased, adequate carbon dioxide (CO_2) elimination may not be possible or higher-than-desired airway pressure might be the only option. Permissive hypercapnia has been promoted as an alternative to increasing V_T or airway pressures, but is unacceptable in patients with increased intracranial pressure or concomitant metabolic acidosis.[4–6] In addition, concerns about long-term neurologic sequelae have prompted efforts for alternatives.

One method of enhancing CO_2 elimination without raising V_T or airway pressure is the flushing of mechanical and anatomic dead space by a secondary independent gas flow at the carina—tracheal gas insufflation (TGI).[7,8] This

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Table 1. Complications/Problems during Tracheal Gas Insufflation

- Altered airway pressures and volume
- Triggering
- Humidification
- Airway trauma/system obstruction
- Monitoring

technique is not new: it was originally proposed as an adjunct to mechanical ventilation in 1969 by Stresemann and Sattler.[9] However, in spite of information regarding the use of TGI available for more than 30 years, there are no commercially available systems to provide TGI.

The primary reason why TGI has not become part of common clinical practice is the complications and problems observed during the use of TGI (Table 1). Many of these problems have simple-to-engineer solutions, but some may be life-threatening if not appropriately monitored and alarmed or if system-interrupt algorithms are not standard. This article discusses the problems inherent in TGI systems and methods to avoid those problems.

Altered Airway Pressures and Volumes

Depending on the TGI system used and the type of TGI catheter placed, an increase or decrease in peak alveolar pressure or end-expiratory pressure may occur during TGI.

Tracheal Gas Insufflation System

Numerous approaches to TGI and a number of different catheter types have been documented in the literature (Table 2). Catheter designs either direct gas flow toward the carina (direct-flow TGI [d-TGI])[10] or toward the ventilator circuit (reverse TGI).[11] TGI delivery systems are either continuous[12] or phasic (expiratory only)[13] and provide either insufflation (TGI)[10–13] or exsufflation (TGE).[14,15,16]

Peak System Pressure

As illustrated in Figure 1, regardless of whether volume-control or pressure-control is used, continuous-flow TGI (c-TGI) increases peak airway pressure, peak carinal pressure, and end-inspiratory pressure, as well as increasing or decreasing total positive end-expiratory pressure (PEEP).[11] In association with these changes is an increase in V_T.[11] During volume-controlled ventilation (see Fig. 1A) the flow from the c-TGI system is additive to the flow from the ventilator. With pressure-controlled ventilation, the addition of the c-TGI flow causes the flow from the ventilator to more rapidly decelerate, and once the ventilator flow reaches zero a square wave flow pattern entirely from the c-TGI system persists. Conceivably, in a patient with a high system compliance, low airways resistance, and short inspiratory time (T_I), inspiratory pressure and V_I might not be altered, but this is highly unlikely. In general, the impact of c-TGI on inspiratory airway pressure is increased: the stiffer the lung, the greater the airways resistance, and the longer the T_I.[11]

Correction of Inspiratory Pressure Changes

A number of different techniques are available to avoid increased inspiratory airway pressure and V_T during TGI. Figure 2 depicts the effects of expiratory TGI (e-TGI) and volume-adjusted TGI (a-TGI).[12] Note that with each of these techniques the inspiratory phase of ventilation is unaffected by the TGI flow.[12] This is always true with e-TGI,[17,18] but only applicable in limited circumstances with a-TGI.[12] First, a-TGI can only be used with volume-controlled ventilation. Second, the interrelation between set V_T, TGI flow, and T_I limits when a-TGI can be used.[12] For example, if V_T is 500 mL and TGI flow is 18 L/min with a T_I of 1.5 seconds, the volume added by the TGI system during inspiration is 450 mL. Thus, setting the ventilator V_T at 50 mL would maintain V_T delivery without pressure change. However, if the V_T were 400 mL instead of 500 mL, it would be impossible to adjust the delivered V_T to compensate for the volume delivered by the TGI system within the 1.5 second T_I.

The use of e-TGI requires interaction of the mechanical ventilator and the TGI system. That is, activation of TGI flow must be coordinated with the onset and termination of the expiratory phase.[12,13,17] Electronic coupling of the TGI system and the ventilator's exhalation valve will accomplish the appropriate interaction, but coordination could

Table 2. Definitions

• TGI	Tracheal gas insufflation
• c-TGI	Continuous-flow tracheal gas insufflation during both inspiration and expiration
• a-TGI	Volume-adjusted continuous-flow tracheal gas insufflation, tidal volume adjusted by TGI volume provided during inspiration
• e-TGI	Expiratory-phase-only tracheal gas insufflation
• d-TGI	Direct tracheal gas insufflation; flow directed toward carina
• r-TGI	Reverse tracheal gas insufflation; flow directed toward ventilator circuit
• TGE	Tracheal gas exsufflation; expiratory phase only

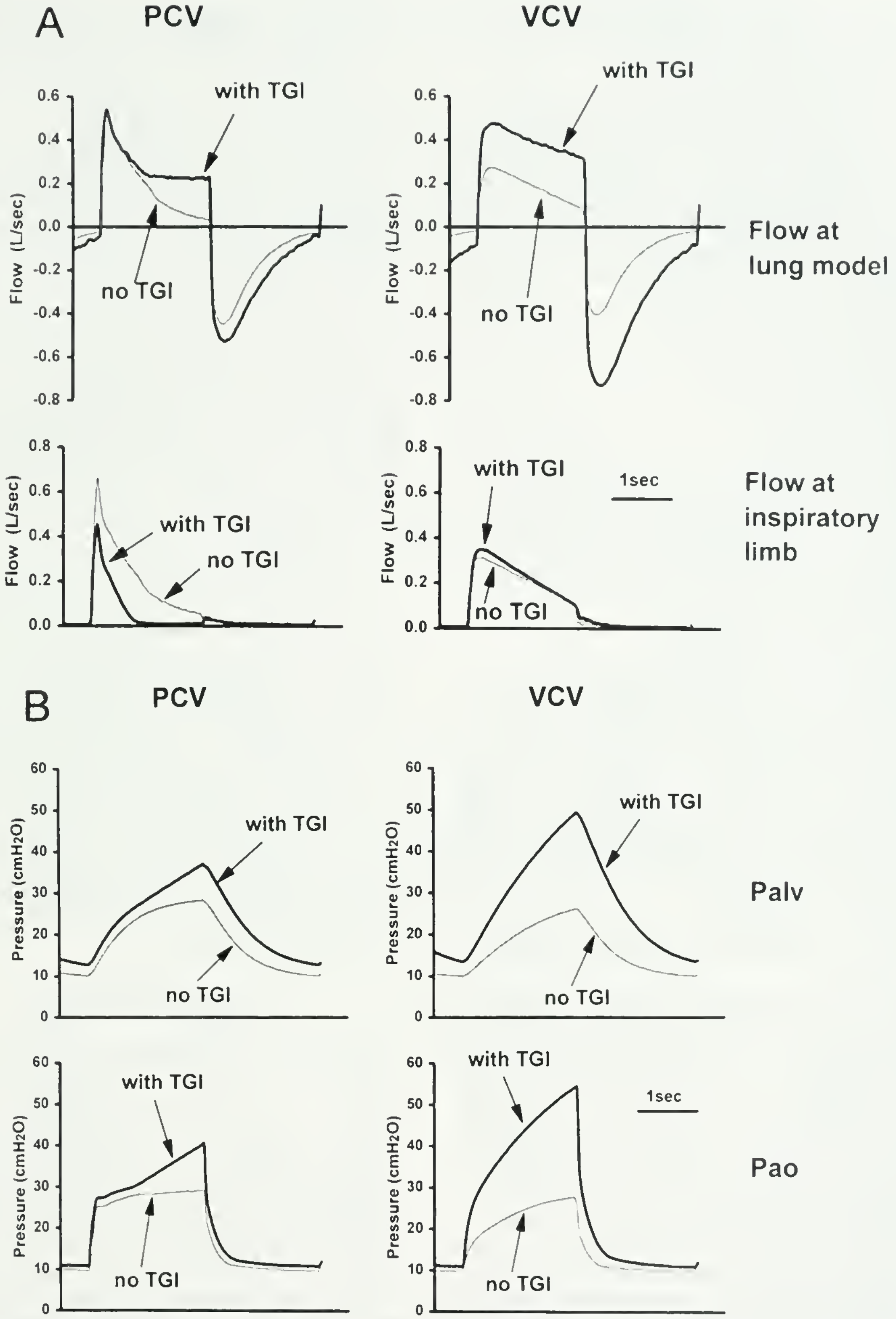

Fig. 1 A: Flow-versus-time tracings of delivered gas flow measured both at airway openings and distal to the entrance of the tracheal gas insufflation (TGI) flow in both pressure-controlled ventilation (PCV) and volume-controlled ventilation (VCV) with and without the addition of 12 L/min TGI flow in a lung model. B: Pressure-versus-time tracings of system pressure measured both at airway opening (PaO) and distal to the entrance of the TGI flow (Palv) in both PCV and VCV, with and without the addition of 12 L/min TGI flow in a lung model. (From Reference 10, with permission.)

also be accomplished by the measurement of flow at the patient airway and activation of the TGI system based on directional changes in delivered flow.

Placement of a pressure relief or flow relief valve into the ventilator circuit would also prevent airway pressure and V_T from increasing during pressure-controlled ventilation.[19,20] Although it is feasible to use these valves during volume-controlled ventilation, it is difficult to ensure constant V_T. The airway pressure waveform tends to look like a pressure-controlled breath, and V_T is no longer the target. When a pressure relief valve is used, it functions most effectively when placed in the inspiratory limb,[19,20] whereas a flow relief valve needs to be placed in the expiratory limb (Edgar Delgado, University of Pittsburgh Medical Center, 2000, personal communication) for maximum effectiveness.

Pressure and volume increases can also be prevented during c-TGI by the use of a ventilator that has an exhalation valve active during inspiration in the pressure-controlled mode.[11,21] The only two ventilators that include this feature at present are the Dräger Evita and the Nellcor Puritan Bennett 840. With an exhalation valve active during inspiration, pressure exceeding the set pressure control level is released to the atmosphere.

Continuous TGI with a pressure relief valve, flow relief valve, or exhalation valve active during inspiration avoids the development of excess system inspiratory pressure and a higher V_T than during pressure-control ventilation (PCV) without TGI. However, the gas flow pattern delivered to the patient will be different from that without the use of TGI.[10] Ventilator flow decreases more rapidly with c-TGI (see Fig. 1B) and the flow pattern during PCV becomes similar to square wave flow, never decreasing to zero flow prior to the end of inspiration.

Alteration in Total Positive End-Expiratory Pressure

During d-TGI, total PEEP is increased (Figs. 3 and 4).[11,12] This occurs regardless of whether TGI is continuous,[11] or during exhalation only,[12] or whether V_T is adjusted (a-TGI).[12] The reason for this is that the TGI flow directed toward the carina acts as a threshold resister. The greater the d-TGI flow, the greater the potential for increased total PEEP. As noted in Figures 3 and 4, lung mechanics affect the development of TGI-induced PEEP, but to a minor extent. The more highly compliant the lung, the greater the TGI-induced PEEP developed. As noted primarily in Figure 3, volume-controlled ventilation results in greater TGI-induced PEEP than pressure-controlled ventilation, because with volume-controlled ventilation and c-TGI, V_T is increased more than with PCV, because there is no mechanism to decrease ventilator-delivered flow, as there is in PCV. In both Figure 3 and Figure 4, T_I appears to affect TGI-induced PEEP, but the increased PEEP associated with increased T_I is a result of both air trapping and intrinsic PEEP (auto-PEEP),[10] and during c-TGI a greater V_T is delivered with the longer T_I. With e-TGI, TGI-induced PEEP is not affected by T_I unless the longer T_I causes air trapping and auto-PEEP.[12,13]

The effects of TGI-induced PEEP can be corrected by careful monitoring of end-inspiratory plateau pressure (P_{plat}) and V_T (Table 3).[10–12] During volume-controlled ventilation the development of TGI-induced PEEP (as with auto-PEEP) causes P_{plat} to increase. Reducing applied PEEP until P_{plat} returns to the level measured before TGI was started restores the total PEEP level to that set before TGI.[12,13] Similarly, during pressure-controlled ventilation the development of TGI-induced PEEP causes V_T to decrease. Reducing the applied PEEP until V_T is restored to the pre-TGI V_T returns total PEEP to the pre-TGI level.[13] During both e-TGI and c-TGI, similar adjustments can be made, but the continuous flow during c-TGI prevents precise measurement of P_{plat} or V_T, preventing accurate corrections.

Reverse-flow TGI (r-TGI) does not result in an increase in total PEEP, but rather a decrease in total PEEP.[11,13] The reverse flow of high-velocity gas creates a jet drag effect at the catheter orifice, decreasing total PEEP. The magnitude of the decrease depends on catheter design and TGI gas flow velocity, not gas flow volume. Systems with large-bore TGI catheters alter total PEEP minutely. In at least one comparison, similar TGI flows resulted in approximately 3.0 cm H_2O PEEP increase with direct flow, but less than 1.0 cm H_2O decrease with reverse-flow.[13]

The decrease in PEEP with r-TGI can be corrected in a manner similar to d-TGI. During volume-controlled ventilation the applied PEEP is increased until the pre e-TGI P_{plat} is restored, whereas during pressure-controlled ventilation the applied PEEP is increased until the pre e-TGI V_T is restored (see Table 3).

Tracheal Gas Exsufflation

By definition, TGE is applied only during the expiratory phase.[14–16] A negative pressure is applied to the exsufflation catheter, removing gas from the lungs. As expected, this does not affect inspiratory pressures and volumes but does decrease PEEP. The effect on PEEP depends on catheter design, catheter exsufflation velocity, and flow. When a similar catheter is used for e-TGI and TGE with the same insufflating and exsufflating velocities, the increase and decrease in PEEP are of similar magnitude.[14] As with r-TGI, the PEEP decrease seen during TGE can be corrected by adjusting applied PEEP (see Table 3). With volume-controlled ventilation, increase applied PEEP until the pre-TGE P_{plat} is reestablished. With pressure-controlled ventilation, adjust applied PEEP until the pre-TGE V_T is reestablished.

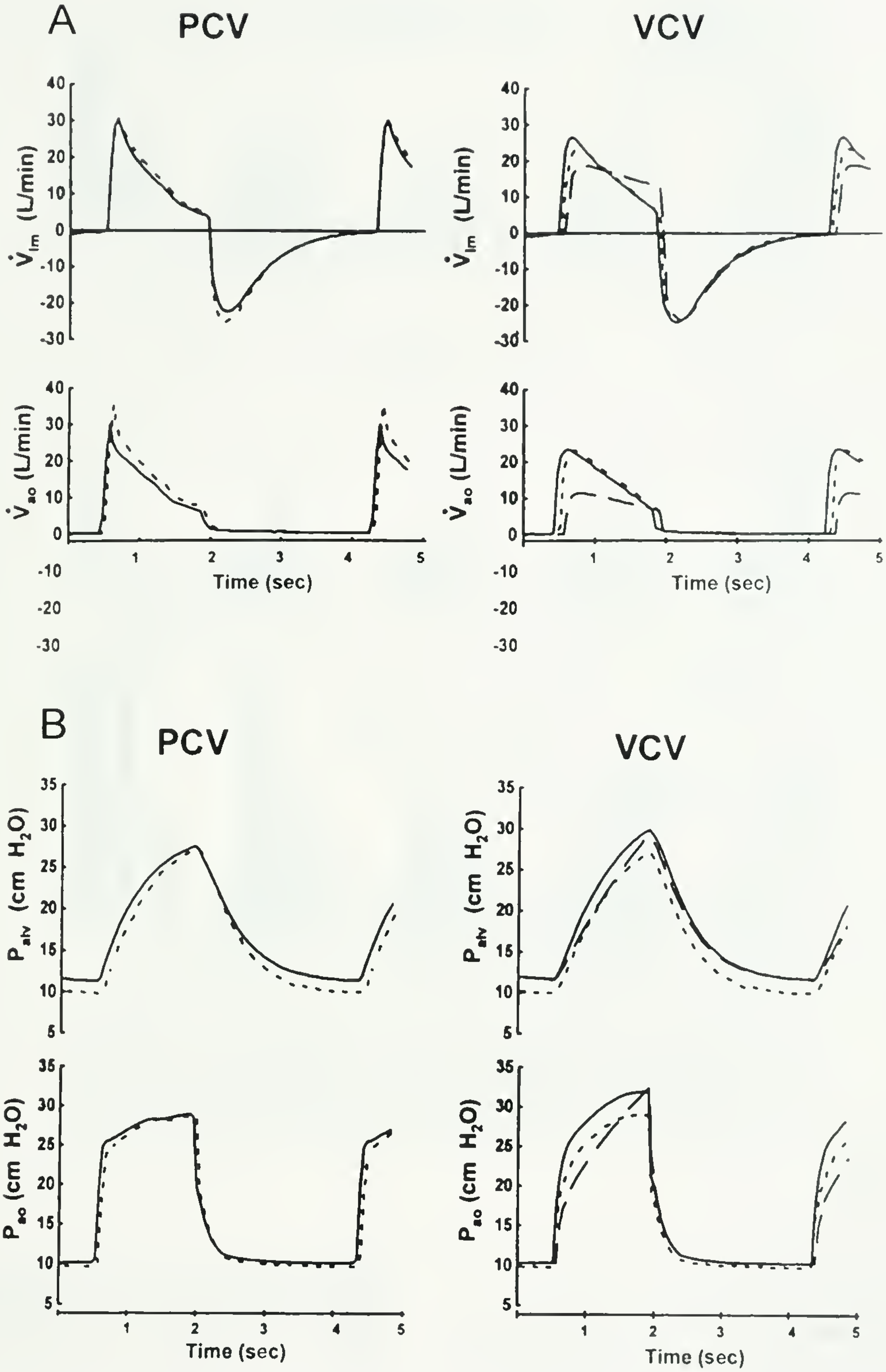

Fig. 2. A: Flow-versus-time tracings of delivered gas flow measured both at the lung model ($\dot{V}_{lm}$) and at airway opening ($\dot{V}_{ao}$) in pressure-controlled ventilation (PCV) (left panels) and volume-controlled ventilation (VCV) (right panels), with and without the addition of tracheal gas insufflation flow. B: Pressure-versus-time tracings of system pressure measured both at the lung model (P_{alv}) and at airway opening (P_{ao}) in PCV (left panels) and VCV (right panels), with and without the addition of tracheal gas insufflation flow. Note that there is considerable overlap among the curves for each tracheal gas insufflation mode. Lung mechanics were set at: compliance = 20 mL/cm H_2O, resistance = 20 cm H_2O/L/s, tracheal gas insufflation flow = 8 L/min, and inspiratory time = 1.5 seconds. (From Reference 12, with permission.)

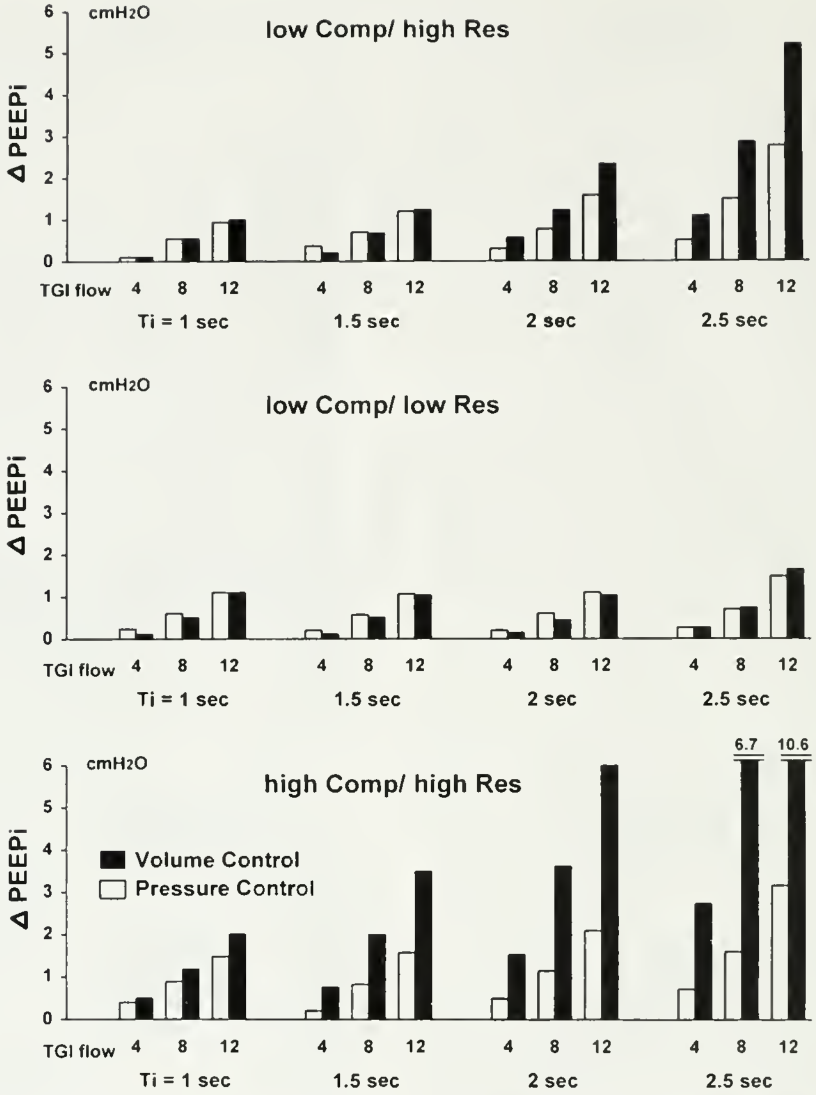

Fig. 3. Changes in total positive end-expiratory pressure (ΔPEEPi) from baseline measurements (zero tracheal gas insufflation flow) at 4 inspiratory times (Ti) for 3 lung mechanics setting in a lung model. Only mean values are listed and all values are $\leq \pm$ 0.5 cm H_2O standard deviation. Tracheal gas insufflation flow values are in L/min. Clear bars indicate pressure-controlled ventilation. Black bars indicate volume-controlled ventilation. Comp = compliance. Res = resistance. (From Reference 10, with permission.)

Triggering

As shown in Figure 5, TGI flow at the end of the expiratory phase may prevent ventilator triggering in certain patients.[22] However, others have shown that adequate gas exchange can be maintained in spite of the TGI flow in patients receiving both continuous positive airway pressure[23] and assisted ventilation.[24] The patients most likely to be affected by the TGI flow are those with diminished ventilatory drive.

The problems associated with triggering can be totally eliminated if the TGI flow is stopped before the end of the expiratory phase or if the TGI flow is coordinated with the ventilator's flow-triggering mechanism.[22] If the ventilator considers the TGI flow in its flow-triggering algorithm, the end-expiratory bias flow from the TGI system can be compensated, and triggering would thus be unaffected.

Reverse TGI or TGE may cause auto-triggering. The sub-baseline end-expiratory pressure created by the TGE system would be expected to trigger the ventilator. As with

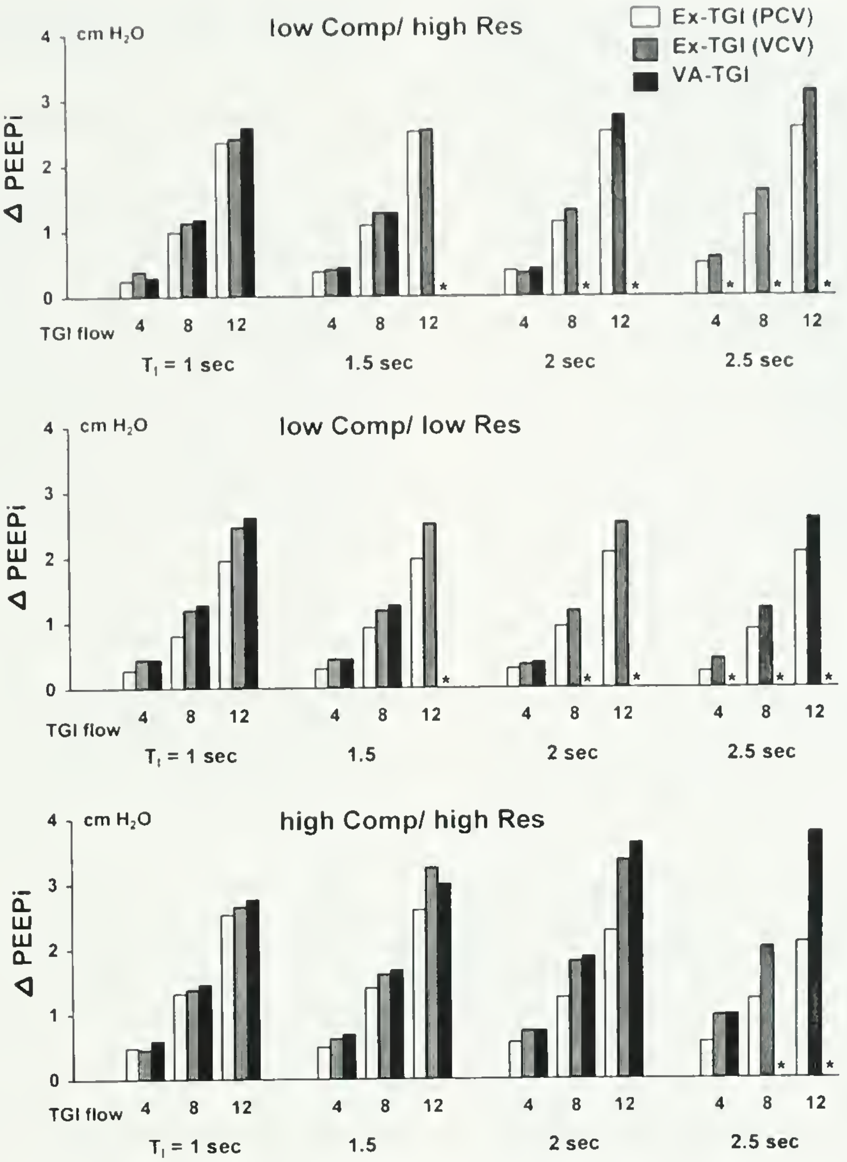

Fig. 4. Changes in total positive end-expiratory pressure (ΔPEEPi) from baseline (zero tracheal gas insufflation flow) at 4 inspiratory times (T_I) for 3 lung mechanics settings. Only the mean values are listed. All values are ± 0.1 (SD) cm H_2O. Comp = compliance. Res = resistance. (From Reference 12, with permission.)

TGI, integration of the TGE system with the ventilator's triggering algorithm would allow for appropriate triggering in spite of the TGE or r-TGI flow.

Humidification

Appropriate humidification of the insufflated gas can be a major problem, especially when d-TGI is used with a very small-bore catheter.[5] Pressures greater than 10 psi may be needed to maintain TGI flow through some catheters.[10] This demands the development of humidification systems capable of tolerating this high pressure. None of the currently available ventilator humidifiers are capable of withstanding this pressure. Humidification systems specifically designed for TGI need to be developed.

Direct-flow or reverse-flow e-TGI minimizes the problems with gas humidification, but a humidifier is still necessary. With TGE no gas is insufflated, and with r-TGI or d-TGI the insufflated flow is primarily moved into the ventilator circuit. However, since r-TGI or d-TGI may

Table 3. Correction of Positive End-Expiratory Pressure Arising from Expiratory-Phase Tracheal Gas Insufflation or Exsufflation

VCV with Direct TGI
- P_{PLAT} increased by TGI-induced PEEP
- Decrease applied PEEP until P_{PLAT} = pre-TGI P_{PLAT}
- Total PEEP = pre-TGI set PEEP

PCV with Direct TGI
- V_T decreased by TGI-induced PEEP
- Decrease applied PEEP until V_T = pre-TGI V_T
- Total PEEP = pre-TGI set PEEP

VCV with Reverse TGI or TGE
- P_{PLAT} decreased by TGI flow
- Increase applied PEEP until P_{PLAT} = pre-TGI P_{PLAT}
- Total PEEP = pre-TGI set PEEP

PCV with Reverse TGI or TGE
- V_T increased by TGI flow
- Increase applied PEEP until V_T = pre-TGI V_T
- Total PEEP = pre-TGI set PEEP

VCV = volume-controlled ventilation
TGI = tracheal gas insufflation
P_{PLAT} = end-inspiratory plateau pressure
PEEP = positive end expiratory pressure
PCV = pressure-controlled ventilation
V_T = tidal volume
TGE = tracheal gas exsufflation

desiccate secretions lining the endotracheal tube, appropriate humidification is required.

Airway Trauma and System Obstruction

Airway trauma is expected primarily when direct-flow catheters that have their distal tips outside the artificial airway are used to deliver TGI. When this occurs, the high-velocity gas flow may be directed to the wall of the trachea, causing erosion of the mucous membrane, as has been observed with jet ventilation.[25] In addition, the farther the catheter tip is from the distal end of the artificial airway, the greater the likelihood that the tip of the catheter will wipe back and forth in the airway, traumatizing the trachea. Large-bore catheters, catheter tips located within the artificial airway, r-TGI, and TGE all minimize the likelihood of tracheal injury.

A major concern with all TGI systems is the development of a mucous ball at the end of the TGI catheter or an obstruction in the artificial airway because of mucosal drying.[26] Either of these situations would be catastrophic if it resulted in complete obstruction of the airway. Regardless of the TGI system or catheter used, it is essential to have a mechanism to identify increased pressure at the

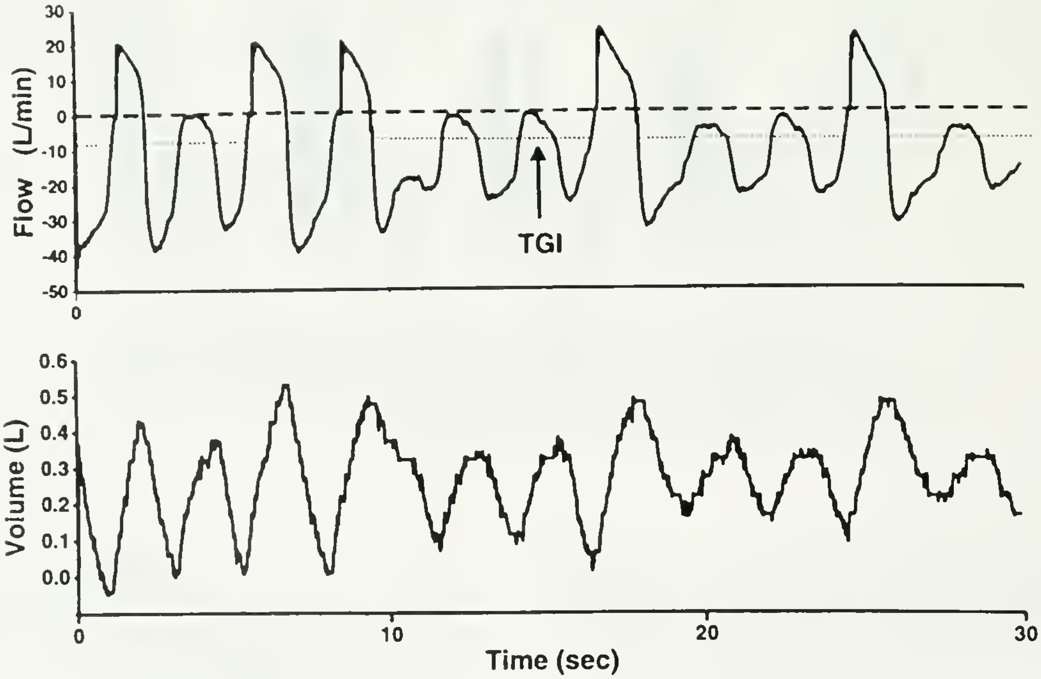

Fig. 5. Flow (top graph) and inductive plethysmographic lung volume tracing (bottom graph) in a mechanically ventilated patient (pressure-support ventilation of 4 cm H_2O) during expiratory tracheal gas insufflation (e-TGI) of 8 L/min. Flow was measured at the Y-adapter (proximal to the catheter orifice), so baseline e-TGI flow was at −8 L/min (dotted line). In this example there are 5 ventilator-delivered pressure-support ventilation breaths; however, there are a total of 11 breaths as measured by the inductive plethysmograph. During these 6 additional breaths, TGI flow met the patient's inspiratory flow demand and provided the total inspiratory volume for the breath and the ventilator was not triggered. (From Reference 22, with permission.)

TGI catheter tip and immediately stop the TGI flow if pressure reaches a threshold.

Monitoring

As is obvious from the previous discussions, careful monitoring of tracheal or TGI catheter pressure is essential. In addition, the ability to measure the P_{plat}, total PEEP, and actual patient V_T and minute ventilation is needed. As a result, the TGI system must either incorporate the ability to perform these measurements or interact with the mechanical ventilator in such a manner as to ensure that these measurements can be made. As is discussed in other papers in this issue of RESPIRATORY CARE, TGI can have a dramatic effect on CO_2 levels, but it can potentially have an equally dramatic negative effect on patients if not properly monitored and controlled.

The Ideal Tracheal Gas Insufflation System

At this stage in the development of TGI it may be premature to describe the ideal TGI system. However, there are clearly a number of features that this system must possess. First, it should be intimately coordinated with the mechanical ventilator. This can be accomplished by a system that talks to the ventilator or is an actual part of the ventilator. Tracheal pressure or catheter pressure monitoring with automatic TGI flow shut-off is essential. The ability to monitor both dynamic and static airway pressures and delivered volume must be present. The TGI system must be integrated into the mechanical ventilator's breath-triggering system to ensure synchrony if used during spontaneous breathing. My bias is that TGI systems should only deliver e-TGI and that r-TGI results in fewer problems than d-TGI. Finally, although it is doubtful that the first commercially developed systems will have the TGI catheter as part of the artificial airway, that would be desirable. The development of artificial airways with large-bore TGI channels reduces the likelihood of airway trauma and reduces the difficulty of humidifying the TGI gas flow.

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Catheters for Tracheal Gas Insufflation

Alexander B Adams MPH RRT FAARC

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Summary

As an adjunct to mechanical ventilation, tracheal gas insufflation (TGI) injects gas flow into the trachea to flush carbon dioxide (CO_2) from the anatomical and mechanical dead space, but the addition of TGI flow from a catheter may cause problems related to increased flow velocity at the catheter tip. Forward momentum and turbulence beyond the tip oppose expiratory flow and may cause or increase intrinsic positive end-expiratory pressure. If the catheter is placed within the endotracheal tube (ETT), the catheter itself acts as a resistive element to exhalation. Effects of the catheter presence (contact on or whipping against the airway) or local rapid gas flow effects on the tracheal mucosa are possible. Thus far, TGI has been delivered through a range of catheter sizes and styles. Two general design modifications have been incorporated in TGI systems to address the possible problems: embedding the catheter flow channel within the ETT, and directing the TGI flow cephalad. The LaBrune-Boussignac tube was designed with 6 or 8 channels embedded within the ETT, from which TGI flow exits laterally within the ETT at 1.5 cm from its tip. This avoids the use of a catheter and thus avoids local traumatic effects. A reverse-thrust catheter has been designed to direct flow within a sheath around the catheter tip; flow exits cephalad from a gap between the sheath and the catheter shaft. As part of a proposed ventilatory mode (intratracheal pulmonary ventilation) the reverse-thrust catheter delivers the tidal breath and, additionally, flushes CO_2 and accelerates secretion removal during exhalation. A reverse-flow design ETT has been developed with two channels, one for tidal volume delivery and the other for TGI flow. The TGI channel is relatively large and flow is directed cephalad by a nozzle at the catheter tip. A recently developed bidirectional catheter allows the option of delivering TGI flow cephalad, towards the lungs or in both directions. Unfortunately, to be convenient, the use of specially designed catheters or ETTs requires the anticipation of TGI use. A complete system for the safe and convenient use of TGI in ventilated patients is not as yet available, but concerns about the safety and convenience of TGI delivery have been addressed with recent advances in catheter/tube design.

Key words: tracheal gas insufflation, catheters, transtracheal oxygen, reverse-thrust catheter, reverse-flow catheter, bidirectional catheter. [Respir Care 2001;46(2):177–184]

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Introduction

The placement of a catheter in the trachea to deliver gas flow was first reported in the late 1960s by Stresemann.[1] The purpose for tracheal gas insufflation (TGI), that is, washout of the anatomical dead space, was clearly described by Stresemann,[1] and, subsequently, Stresemann tested TGI in an animal study and in two ventilated patients.[2,3] In the reported patient cases, size #16 gauge catheters were positioned in the tracheas of the ventilated patients, and TGI was shown to successfully flush more than the anatomical dead space. Using TGI for carbon dioxide (CO_2) elimination was not reported again until Bergofsky and Hurewitz delivered TGI to spontaneously breathing patients 20 years later.[4,5] These investigators applied "airway insufflation" through transtracheal oxygenation catheters (SCOOP, Transtracheal Systems, Denver, Colorado) to spontaneously breathing, hypercapnic patients. Further progress in TGI catheter development has occurred within the past 10 years, although a method of conveying TGI flow to the tracheal region has not, as yet, been standardized. Several issues remain concerning the safety and convenience of delivering TGI flow. The development of catheters or tube designs for TGI has attempted to address several concerns about safety and convenience, as well as to determine TGI's effectiveness. Recent development has been rapid: several innovative catheters and tubes have been designed and tested in bench settings. Less experience has been reported, thus far, from the clinical setting. Without directly discussing the effectiveness of CO_2 elimination by TGI with various catheters, the physical characteristics and general purposes of the TGI catheters/tubes are described herein.

Technical Considerations

There have now been several decades of experience with ventilator circuitry. Though the dynamics of air flow throughout the airways continues to be crudely understood, the air flow dynamics of the ventilator circuitry are more easily studied and better understood. With the integrated use of a relatively fixed structure such as an endotracheal tube (ETT) or a TGI catheter, some basic concepts can be assumed to apply to TGI. The important concepts of TGI delivery are associated with driving gas flow into a narrow TGI flow channel(s). As the gas flow channel narrows into the TGI catheter, velocity increases in direct proportion to the reduction in channel area (Fig. 1). At the same time, to drive gas flow through the catheter, back pressure will develop in the tubing or chambers (such as humidifiers) prior to the narrowed lumen (Fig. 2). Even more importantly, when the gas exits the TGI channel with increased velocity, the forward flow momentum is directed toward the lungs. Also, beyond this exit point the gas emitting from the catheter tip causes markedly increased turbulence. Then, during the patient's expiratory phase, the forward momentum of lung-directed TGI flow and the increased turbulence tend to oppose expiratory flow. This opposing flow can cause intrinsic positive end-expiratory pressure (auto-PEEP) or increased end-expiratory volume. Another factor is the additional flow from the TGI catheter introduced into the airway during exhalation. This additional flow can increase the role of the ETT and exhalation valve of the ventilator as resistive elements. Also, the physical presence of a catheter in the airway may become a factor during exhalation. The presence of a TGI catheter decreases the lumen area within the ETT and may act as a resistive element that impedes expiratory flow. This effect could be important with a larger-diameter TGI catheter and an ETT with a smaller internal diameter. Though a smaller TGI catheter could reduce this effect, a smaller TGI catheter tends to whip at higher flow rates. With a smaller catheter, air stream velocity is even higher, and the gas stream may become directed at the airway mucosa. Also, unless the increased flow is heated and humidified, a drier, cooler TGI gas may be directed at the airway mucosa. These local effects due to turbulence of the TGI stream or the catheter (whip, stream effect, cooling, drying) can be called local jet effects. Finally, depending on the phasing of TGI flow and pressure sensing devices in the ventilator or circuitry, lung pressure generation beyond the TGI catheter tip is possible if the ETT narrows or occludes. Therefore, concerns about catheters delivering TGI flow in the trachea have included:

1. Upstream pressure buildup in the humidifier and the external TGI circuitry.
2. Post-catheter effects on exhalation caused by turbulence and momentum transfer.
3. Additional resistance of the ETT and exhalation valve with the addition of TGI flow.
4. Catheter presence serving as a resistive element during exhalation.
5. Local jet effects on the airway due to:
   a. Catheter whip
   b. High-velocity air stream

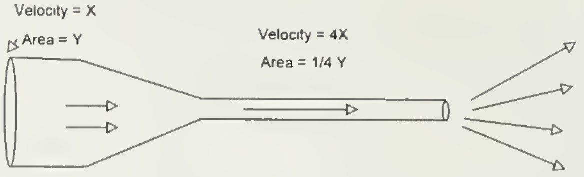

Fig. 1. The effect of driving a fixed gas flow through a narrowed lumen. 1. Back pressure generates prior to the narrow lumen. 2. Flow velocity increases. 3. Turbulence occurs at the lumen exit port. 4. Forward momentum results from the high gas velocity at the exit port.

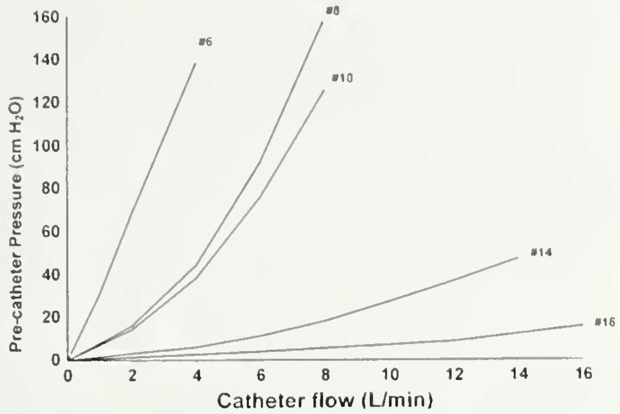

Fig. 2. Back pressure generates upstream from a tracheal gas insufflation catheter. This pressure generation is in an exponential relationship to increasing flow and is directly related to decreasing catheter size. (Adapted from Adams AB, Tracheal gas insufflation. Respir Care 1996;41[4]285–291.)

c. Cooling and drying of the airway

6. Pressure generation in the lungs with TGI flow occluded or impeded exhalation.

Several innovative catheters and tubes have been developed to address these possible problems. There have been two major design solutions to these potential problems:

1. The catheter itself can be removed from the ventilator circuit by delivering TGI via a channel(s) in a specialized ETT.

2. The use of a catheter designed to direct flow away from the lungs (cephalad).

In both solutions, the need for the TGI tube or catheter must be anticipated—the specialized tube should be used for intubation or the catheter must be obtained. Therefore, these two possible solutions may be effective at reducing undesired effects of TGI, but their use may not be convenient.

Catheters and Tubes

Transtracheal Oxygenation

In spontaneously breathing patients, extensive experience has been gained in the use of catheters emitting flow into the tracheal region. Transtracheal oxygen (TTO) catheters have been used for oxygen supplementation flow since the 1980s. TTO is a sophisticated form of oxygen therapy, with a different purpose than TGI, yet the introduction of gas flow via a catheter is not, functionally, very different between the two techniques (they are both TGI). The experience with TTO provides evidence that a catheter positioned in the trachea can be safely maintained over extended periods without serious complications.[5,9] The most common complications reported with TTO have been small mucus balls in proportion to the catheter size in use, the catheter clogging or becoming dislodged, and increased phlegm production. Though the stoma and catheter must receive meticulous care with the use of TTO, the experience from TTO suggests that TGI may not have important complications from local jet effects (or other effects?) on the airways and lungs.

Miscellaneous Catheters

The initiation of TGI in the clinical setting can be dangerously simple. Technically, the only extra equipment required for adding TGI to the ventilator circuit would be a flowmeter, oxygen tubing, a bronchoscope adapter, a catheter-oxygen tubing connector, and the catheter (Fig. 3). The flowmeter with oxygen tubing is attached to the connector and catheter, a bronchoscopy adapter is inserted at the end of the ETT, and the TGI catheter can then be positioned. As an adjunct to mechanical ventilation, only prone positioning is simpler and less expensive. But with the indiscriminate use of a simple TGI circuit, all catheter-related problems can occur. Many catheter sizes and types have been used for TGI (Table 1). Recommendations for

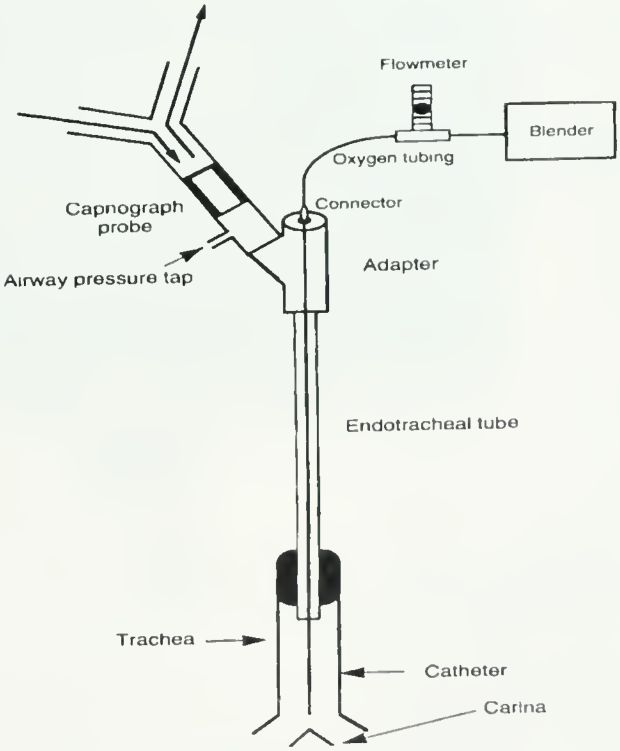

Fig. 3. Typical schematic of a simple tracheal gas insufflation (TGI) circuit: a gas flow source (flowmeter), a blender to delivery fraction of inspired oxygen (F_{IO_2})-matched gas, oxygen tubing, connector, bronchoscopy adapter, and the TGI catheter (Adapted from Adams AB, Tracheal gas insufflation. Respir Care 1996;41[4] 285–291.)

Table 1. Sizing of Channels for Tracheal Gas Insufflation

Study	Internal Diameter (mm)	Catheter Size
Isabey[10]	1.0	
Heimlich and Carr[6]	1.7	5 French,
Miro et al[11]		16 gauge
Imanaka et al[12]		
Nakos et al[13]	2.0	
Bergofsky and Hurewitz[4]	2.1	14 gauge
Hurewitz et al[5]		
Ravenscraft et al[14]		7 French
Kolobow et al (reverse thrust catheter)[15]	2.64	
Christopher et al[7]		8 French
Kirmse et al (reverse flow design)[16]	3.54	

the selection of TGI catheter size and type have not been published. Clinical studies of TGI have used this simple TGI catheter circuit, but these studies have included additional monitoring and the attention of data collection.[13,14,17]

Specialized Catheters

Several specialized catheters have been used to study specific questions about TGI or to study more general issues of ventilation. For example, Slutsky and Menon conducted an animal study of constant-flow (apnea) ventilation using a specialized double-lumen catheter.[18] The catheter was placed alongside, not through, the ETT (Fig. 4). With the two catheters placed outside of the ETT, they did not act as resistive elements, and the ETT served as a conduit for exhalation only. The catheters were positioned sequentially at several distances beyond the carina while continuous gas flow was delivered at high flows without inspiratory/expiratory cycling (apnea ventilation). That study reported adequate blood gases with high constant-flow ventilation via airway-delivered gas flow.

In two other animal studies, TGI was delivered via a well-secured, carefully positioned small-lumen metal tube.[19,20] The metal catheter provided a fixed directionality for TGI flow, eliminating the possibility of catheter whip or malpositioning.

Another study delivered TGI through an ETT designed for high-frequency ventilation (Mallinckrodt Hi-Lo jet tube),[21] which has two channels tunneled within the ETT, one for jet ventilation and one for pressure monitoring.The pressure monitoring channel was used for TGI delivery to allow convenient application of phasic-expiratory TGI and avoid the use of a catheter.

In a recent study, Blanch et al reported the use of a Univent (Phycon) catheter in a unilateral lung injury model.[22] This catheter was required to aim the therapy (TGI) at the region of injury—the injured lung. Injury-directed TGI was remarkably effective at CO_2 elimination. In each of the cited studies, specialized catheters were used, primarily, for research purposes. These catheters/tubes were not being studied or tested for possible clinical use.

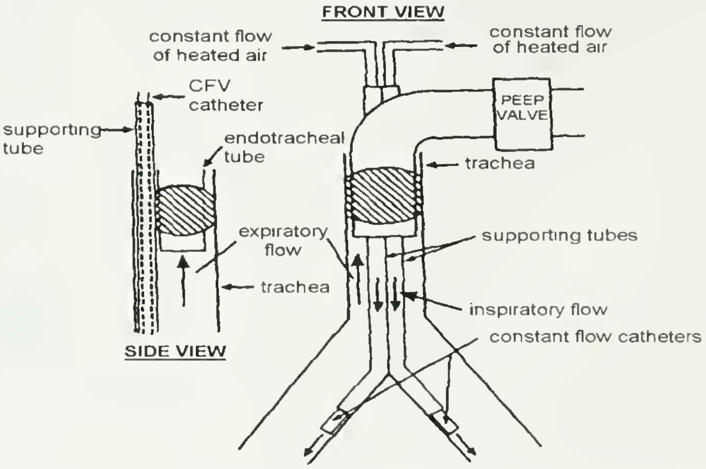

Fig. 4. Schematic of a double-lumen tracheal gas insufflation (TGI) catheter system used in a study of constant-flow (apnea) ventilation. The two catheters are alongside and in back of the endotracheal tube (ETT). Constant gas flow is delivered by the catheters, with gas exiting the lungs via the ETT. The study protocol involved the positioning of catheters at various distances into the airways beyond the ETT. CFV = constant flow ventilation. PEEP = positive end-expiratory pressure. (From Reference 18, with permission.)

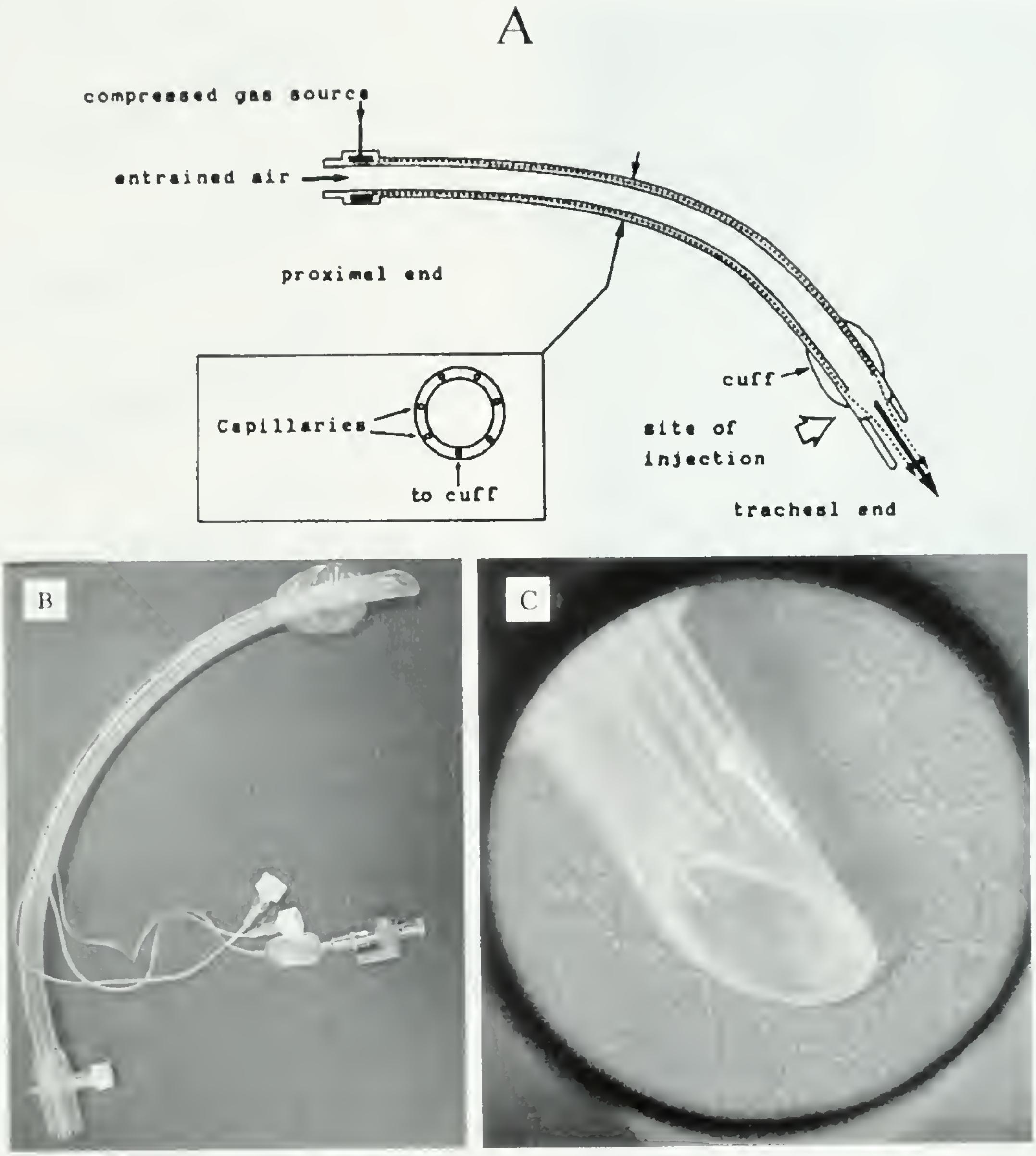

Fig. 5. The LaBrune-Boussignac endotracheal tube (ETT). A: Schematic of the entire tube, with a mid-tube cross-section. B: Photograph of the tube. C: Photograph of the tube tip. (From Reference 10, with permission.)

The LaBrune-Boussignac Tube

A specialized ETT, the LaBrune-Boussignac tube, has been developed in France, with an original purpose of providing a means to bypass ETT resistance. It was also considered as an option for delivering constant-flow ventilation (Fig. 5).[10] This tube has now been studied as a TGI device.[23,24] The tube has either 6 or 8 channels molded along the length of its inner shaft. Gas entry into the ETT is via a manifold at the top of the tube, and the channels direct TGI flow to a lateral entry within the ETT at 1.5 cm from the tip. One of the ports is reserved for tracheal pressure monitoring, and another is available for the option of instilling surfactant. This tube was studied in premature newborns as a TGI device during mechanical ventilation, and its use allowed a less aggressive conventional ventilation strategy.[23,24] There are several possible benefits to the use of the LaBrune-Boussignac tube. The use of multiple channels distributes gas flow and reduces the velocity of TGI gas. With exit ports inside the tube, local jet effects are reduced and not directed upon the airway. Embedding the TGI channels into the ETT avoids, of course, the need for a TGI catheter.

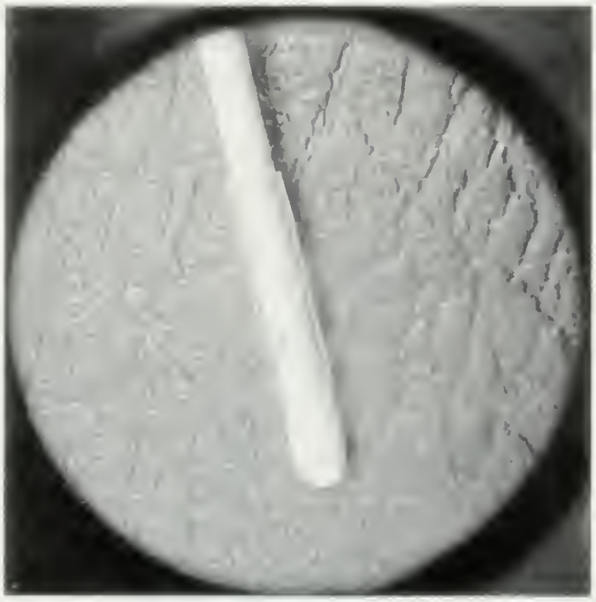

Fig. 6. A reverse-thrust catheter. This catheter has a closed end with side ports for the exiting TGI flow. Flow is directed cephalad along a gap between a sheath and the catheter. (From Reference 15, with permission.)

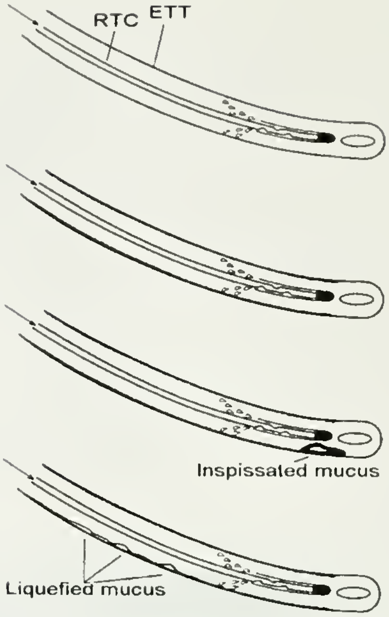

Fig. 7. Secretion removal by the reverse-thrust catheter (RTC). This schematic illustrates the potential role for movement or jetting of liquid secretions and inspissated secretions toward the mouth. ETT = endotracheal tube. (From Reference 26, with permission.)

Reverse-Thrust Catheter

A specially designed TGI catheter has been developed as part of a ventilation method: intratracheal pulmonary ventilation.[15,25] This catheter has an occluded tip, with catheter flow exiting via side ports. The TGI flow is further directed cephalad along an annular gap (Fig. 6). This is called a reverse-thrust catheter (RTC) because it directs TGI flow away from the lungs. With an RTC, the local jet effects from TGI flow are within the ETT, and TGI flow is aimed away from the lungs, in the direction of (not opposing) expiratory flow. The RTC design may then have another possible advantage: a higher-velocity TGI flow will not impede expiration if a Venturi effect by the RTC flow entrains rather than impedes flow from the expiratory flow stream. In intratracheal pulmonary ventilation, the RTC also delivers the tidal volume. With the RTC flow running continuously, inspiration occurs when the exhalation valve of the ventilator closes and tidal volume is delivered to the lungs via the RTC. When the expiratory valve opens (during exhalation) the ETT serves (as its primary role) as the conduit for exhalation. As experience with the device has accumulated, an observation has led to another possible beneficial use of the RTC. The RTC accelerates secretion removal from the ETT and airways, so RTC assists with airway clearance (Fig. 7).[26] RTC use may therefore avoid or reduce the need for intermittent endotracheal suctioning.

Reverse-Flow-Design Tube

A double-lumen ETT using a reverse-flow design (RFD) tube (Fig. 8) has been devised for use with TGI.[16,27,28] The larger of the two lumens delivers the tidal breath to the patient. A smaller lumen delivers the TGI flow. At the end of the TGI lumen a nozzle redirects the TGI flow cephalad (as with the RTC). Again, incorporating the TGI flow within the ETT and directing TGI flow cephalad has some advantages. Of course, with the RFD ETT (as with the LaBrune-Boussignac tube), a catheter is not required. Local jet effects are contained within the ETT. And, as with the RTC, air entrainment above the nozzle by the TGI flow can assist rather than impede expiratory flow.

The advantages of the LaBrune-Boussignac, RFD, and RTC tubes/catheter can be considered improvements in the delivery of TGI. Unfortunately, with use of the LaBrune-Boussignac or RFD tubes, the use of TGI should be anticipated prior to intubation. Otherwise, starting TGI requires extubation and immediate reintubation with a TGI ETT, a possibly unsafe procedure in a critically ill patient. For the RTC to be used, the catheter must be obtained. Furthermore, its use has been primarily but not exclusively reported with intratracheal pulmonary ventilation, a mode unfamiliar to most clinicians. Therefore, without the ability to anticipate the need for TGI, the application of TGI may continue to be either unsafe or inconvenient.

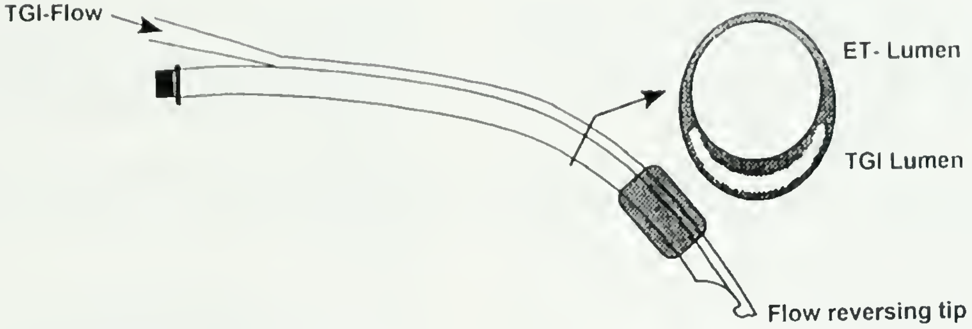

Fig. 8. The reverse-flow design endotracheal tube. A single relatively large channel is embedded in the endotracheal tube. A nozzle at the tip of the tube directs the tracheal gas insufflation flow cephalad. (From Reference 28, with permission.)

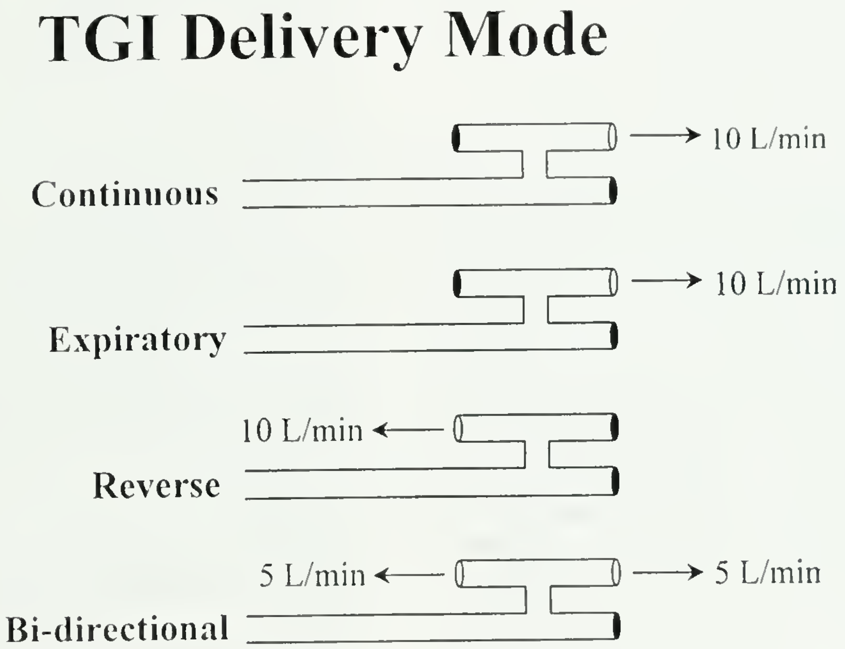

Fig. 9. Schematic of a bidirectional tracheal gas insufflation (TGI) catheter. TGI flow direction can be chosen with this catheter, either toward the lungs (distal), toward the mouth (proximal), or in both directions. Expiratory or continuous TGI can be delivered. (Figure courtesy of Edgar Delgado.)

A Bidirectional Catheter

A catheter with an H-shaped tip was recently introduced that also addresses the issue of TGI-induced auto-PEEP generation (Fig. 9).[29] This catheter allows control of the direction of TGI flow. Flow can be directed toward the lungs, in a reverse flow (cephalad), or in both directions at once. Though the lung-directed flow may induce auto-PEEP, the flow may have the benefit of CO_2-clearing effects beyond the catheter. The reverse-flow option has been shown to reduce auto-PEEP in a test lung study.[29] This catheter may allow a balancing of CO_2-elimination effect, local jet effects, and auto-PEEP generation.

Summary

A number of catheter/tube models have been designed to address the concerns of adding TGI flow to the trachea of a ventilated patient. While the role and effectiveness of TGI as an adjunct to mechanical ventilation becomes clearer, to gain wider acceptance a TGI catheter or catheter system must also be shown to be safe and relatively convenient to use. Recent advances in catheter/tube design have addressed concerns regarding the safety and convenience of TGI delivery.

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Monitoring and Humidification during Tracheal Gas Insufflation

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and Michael R Pinsky MD

Introduction
Monitoring
  Tidal Volume
  Peak Inspiratory Pressure
  Intrinsic Positive End-Expiratory Pressure
  Carbon Dioxide Elimination Efficiency
Humidification
Summary

In order to use tracheal gas insufflation (TGI) in a safe and effective manner, it is important to understand potential interactions between TGI and the mechanical ventilator that may impact upon gas delivery and carbon dioxide (CO_2) elimination. Furthermore, potentially serious complications secondary to insufflation of cool, dry gas directly into the airway and the possibility of tube occlusion must be considered during use of this adjunct modality to mechanical ventilation. Regardless of the delivery modality (continuous TGI, expiratory TGI, reverse TGI, or bidirectional TGI), conventional respiratory monitoring is required. However, TGI with mechanical ventilation can alter tidal volume and peak inspiratory pressure and can lead to the development of intrinsic positive end-expiratory pressure. Therefore, depending on the gas delivery technique used, it is important to carefully monitor these ventilatory parameters for TGI-induced changes and understand the potential need for adjustments to ventilator settings to facilitate therapy and avoid problems. Optimally, gas insufflated by the TGI catheter should be conditioned by addition of heat and humidity to prevent mucus plug formation and potential damage to the tracheal mucosa. Finally, patients must be closely monitored for increases in peak inspiratory pressure from obstruction of the tracheal tube and should have the TGI catheter removed and inspected every 8–12 hours to assess for plugs.

Key words: tracheal gas insufflation, monitoring, humidification, mucus plug. [Respir Care 2001;46(2): 185–192]

Introduction

The process of insufflating fresh gas directly into the trachea to augment gas exchange has been utilized for more than 15 years, in spontaneously breathing patients, as transtracheal oxygen delivery.[1–4] More recently this adjunctive technique has been used in conjunction with volume-controlled or pressure-controlled ventilation and is

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referred to as tracheal gas insufflation (TGI).[5,22] The major effect of TGI is to enhance gas exchange efficiency by removal of carbon dioxide (CO_2) from the anatomic dead space.[5–7] When used in conjunction with mechanical ventilation, TGI may alter other parameters that affect CO_2 elimination, such as tidal volume (V_T) and peak inspiratory pressure (PIP), and can lead to the development of intrinsic positive end-expiratory pressure (auto-PEEP).[7,9,10,12–14,20–24] A thorough understanding of interactions between TGI and these ventilatory parameters is critical to utilizing TGI in a safe, effective, and efficient manner. It is equally important to understand how to monitor for adverse effects that may result from these interactions. To avoid potential complications caused by insufflation of cool, dry gas directly into the airway, humidification must also be considered when applying TGI clinically.[25–29] This article reviews issues related to monitoring and humidification when using TGI and suggests possible methods to minimize and/or prevent adverse effects when using this adjunctive therapy.

Monitoring

The statement "appearances can be deceiving" is very applicable to the subject of monitoring during use of TGI. Based on early descriptions, TGI appeared to be a relatively simple therapy. However, complexities involved in its use became evident as utilization of this therapy increased. Authors have described a variety of interactions between TGI and mechanical ventilation that influence V_T, minute ventilation ($\dot{V}_E$), airway pressure, and/or total PEEP (ventilator set PEEP + auto-PEEP).[9–13,20–23] These interactions necessitate specific adjustments and patient/ventilator monitoring (Table 1).

Tidal Volume

Numerous studies have shown that continuous TGI (c-TGI) causes an increase in inspired V_T when used in conjunction with mechanical ventilation.[9–11,21,23] This phenomenon is addressed in various ways, depending on the ventilatory mode and TGI delivery method.

During volume-controlled ventilation, the total V_T delivered is equivalent to the ventilator set V_T plus the V_T generated by the TGI catheter. That is: $V_{Ttotal} = V_{Tvent} + V_{Tcath}$.

The additional volume delivered by the catheter can be determined by calculating the amount of TGI-generated V_T and subtracting this value from the ventilator-set V_T. With this adjustment a consistent V_T is delivered to the patient prior to and following initiation of TGI. For example, consider a patient on assist-control ventilation with a set V_T of 700 mL, respiratory rate of 15 breaths per minute, and inspiratory time (T_I) of 1.5 seconds. If TGI is initiated at 6 L/min, the V_T derived from TGI is calculated as follows:

$$V_{T\,TGI} = (V_{TGI}\ \text{L/min})(T_I)(1\ \text{min}/60\ \text{s})(1{,}000\ \text{mL/L})$$

$$= 150\ \text{mL}$$

where V_{TGI} = TGI flow in L/min, T_I = inspiratory time in seconds, the calculation 1 min/60 s converts from seconds to minutes, and the calculation 1,000 mL/L converts from liters to milliliters.

The TGI-derived V_T (150 mL) is then subtracted from the ventilator set V_T to obtain the adjusted ventilator V_T setting. Thus, in this example:

$$550\ \text{mL}(\text{adjusted } V_T)$$

$$= 700\ \text{mL (set } V_T) - 150\ \text{mL } (V_{T\,TGI})$$

This V_T adjustment prevents an increase in the total delivered V_T during TGI.

During pressure-controlled ventilation (PCV) and c-TGI, the ventilator and catheter function together to deliver gas over the preset T_I. As the catheter delivers gas, ventilator flow decreases because the catheter's added gas contributes to achieving the set inspiratory pressure.[25] Under certain conditions (eg, c-TGI flows ≥ 10 L/min, long T_I, low resistance), set inspiratory pressure may be reached before the end of inspiration. Accordingly, flow from the ventilator ceases and the expiratory valve remains closed until the end of inspiration. However, the catheter continues to deliver gas into the lung, resulting in an increase in delivered V_T. This problem can be easily corrected by inserting a pressure relief valve (Bird, #04230, Bird Products, Palm Springs, California) into the ventilator circuit, allowing TGI flow (excess volume) to be vented into the atmosphere (Fig. 1).[23] If set inspiratory pressure is changed, adjustments must be made to the pressure relief valve. Delgado et al recently tested a prototype flow relief valve (Respironics, Murrysville, Pennsylvania) that removes gas from the circuit at a set flow (eg, 10 L/min) (Fig. 2).[30] Use of this valve may simplify monitoring during TGI administration because it eliminates the need to use a pressure relief valve and make adjustments to the pressure relief valve if set inspiratory pressure is changed.

Use of expiratory TGI (e-TGI) during volume-control ventilation prevents the delivery of additional inspired V_T because gas flow is only activated during expiration. However, during PCV, the volume of gas delivered to the airway depends on the pressure gradient between peak intrapulmonary pressure and end-expiratory lung pressure. If this pressure gradient decreases because of the development of auto-PEEP, the V_T delivered to the patient will

Table 1 Monitoring and Ventilator Adjustments during Tracheal Gas Insufflation

Variable	Ventilatory Mode	TGI Delivery Mode	Action
Tidal volume	VCV	Continuous	Reduce inspired V_T to compensate for TGI flow during inspiration (see text for calculation) or insert a flow-relief valve
		Expiratory	No modification required
		Reverse	Reduce inspired V_T to compensate for TGI flow during inspiration (see text for calculation) or insert a flow-relief valve
		Bidirectional	No modification required if flow-relief valve is used
Airway Pressure	PCV	Continuous	Insert pressure or flow-relief valve
		Expiratory	May need to increase set inspiratory pressure to maintain V_T
		Reverse	May need to increase set inspiratory pressure to maintain V_T
		Bidirectional	No modification required if flow-relief valve is used
Auto-PEEP	VCV	Continuous	Reduce ventilator PEEP if PIP increases. Clinically, a 5 cm H_2O reduction in set PEEP is typically required @ TGI flows of 10 L/min
		Expiratory	Same as with continuous
		Reverse	An increase in ventilator PEEP may be necessary if NEEP is generated or total PEEP is decreased
		Bidirectional	No adjustment necessary
Auto-PEEP	PCV	Continuous	Same as with VCV
		Expiratory	Same as with VCV
		Reverse	Same as with VCV
		Bidirectional	No adjustment necessary

TGI = tracheal gas insufflation. VCV = volume-control ventilation. V_T = tidal volume. PCV = pressure-control ventilation. auto-PEEP = intrinsic positive end-expiratory pressure. PIP = peak inspiratory pressure. NEEP = negative end-expiratory pressure.

decrease.[22,24] To maintain a constant V_T it is therefore necessary to increase set inspiratory pressure by an amount equivalent to the amount of auto-PEEP generated, or decrease set PEEP by the amount of auto-PEEP generated.

With reverse TGI (r-TGI), other monitoring issues may arise. Several studies have examined interactions between volume and pressure when using r-TGI alone and in combination with e-TGI.[14,16–18] In those studies, r-TGI produced equivalent PEEP, compared with conventional ventilation at low flows, but negative end-expiratory pressure was generated when $\dot{V}_E$ was increased (double or tripled) for 10–20 seconds.[16] Decreases in total-PEEP were also generated when r-TGI was delivered only during expiration at 10 L/min, subsequently reducing lung volume.[14] These observations are important when monitoring patients receiving TGI, because the V_T delivered during PCV depends on the pressure gradient between peak intrapulmonary pressure and end-expiratory lung pressure.

Peak Inspiratory Pressure

Typically, PIP and V_T exhibit a direct relationship when compliance and resistance are unchanged. As V_T is increased, PIP increases. Conversely, as V_T is decreased, PIP decreases. Therefore, the changes in PIP that occur when using a ventilator mode in conjunction with a specific TGI delivery method will be directly related to those observed in V_T

Intrinsic Positive End-Expiratory Pressure

Though there is controversy regarding the impact of different TGI delivery methods on increases in total PEEP, there is consensus that TGI often creates auto-PEEP.[7,9,10,12,14,20–24] Miro et al[20] proposed a theoretical framework to depict the interactions between TGI and ventilator mode, V_T, and PIP that result in the development of auto-PEEP (Fig. 3). During volume-controlled ventilation, when ventilator PEEP is left constant, V_T remains constant, but there is an increase in end-expiratory pressure and, therefore, peak airway pressure because of TGI-induced auto-PEEP. During PCV, when ventilator PEEP and peak airway pressure are kept the same as baseline, V_T excursions (and hence $\dot{V}_E$) are reduced because of TGI-induced auto-PEEP. When ventilator PEEP is reduced by an amount equivalent to TGI-induced auto-PEEP, V_T, peak airway pressure, and total PEEP remain the same as baseline during PCV. This conceptual framework demonstrates the impact of TGI on these ventilatory parameters, and the importance of monitoring for the development of auto-PEEP during TGI administration.

Several mechanisms may contribute to an increase in auto-PEEP when using TGI. The TGI catheter decreases

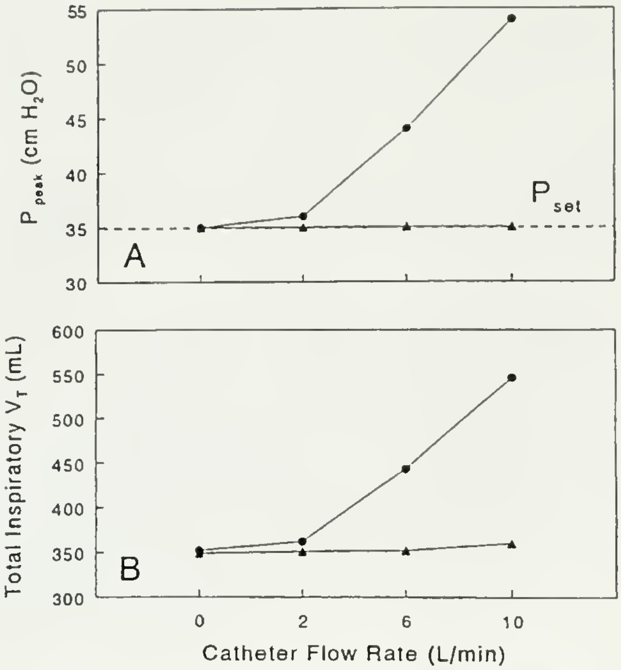

Fig. 1. Effects of catheter flow rate with (circles) and without (triangles) the pressure relief valve on (A) peak airway pressure (P_{peak}) and (B) total inspiratory tidal volume (V_T). Independent variables were constant at airway resistance = 20 cm H_2O/L/s, lung compliance = 0.01 L/cm H_2O, ventilator frequency = 10 breaths/min, and ratio of inspiratory time to total breathing-cycle time = 0.33. The horizontal dotted line at 35 cm H_2O represents the set inspiratory pressure (P_{set}). (From Reference 23, with permission.)

airway cross-sectional area and thereby increases expiratory resistance. The magnitude of this change is proportional to the outer diameter of the catheter. From this perspective, the "optimal" TGI catheter would have a small outer diameter or be incorporated into the endotracheal tube in a manner that does not reduce the inner diameter of the tube. Several studies have tested the efficacy of TGI delivered through a double-lumen endotracheal tube[13,14] or an endotracheal tube that incorporates fine capillaries molded into the lumen.[15] No problems were noted with use of these tubes. However, a major disadvantage is the need to use a special endotracheal tube in all patients who might need this therapy or the need to replace a conventional tube with a special tube in a compromised patient.

A second factor that can increase auto-PEEP is the amount of time dedicated to expiration. The expiratory phase is particularly critical during TGI administration. Expiratory time is inversely proportional to the amount of total PEEP generated, regardless of the method used to deliver TGI.[21,23] Also, the majority of CO_2 washout during TGI occurs during expiration.[10] Therefore, it is important to consider the impact of the inspiratory-expiratory ratio on the development of auto-PEEP.

A third factor to be considered is catheter configuration. Stagnation pressure (back pressure) can develop as forward flow from a straight-tip TGI catheter meets the opposing gas flow exiting the lung.[7] Early studies postulated that the increase in back pressure (and, thus, auto-PEEP) was greater during c-TGI than during e-TGI because c-TGI delivers gas throughout the respiratory cycle.[10,21] However, we have found that when $\dot{V}_E$ is maintained constant, c-TGI and e-TGI produce equivalent levels of total PEEP when delivered with a straight-tip catheter.[24]

Alternatively, TGI can be delivered with a reverse tip (reverse thrust) catheter or a catheter that delivers bidirectional flow (bi-TGI). With bi-TGI, gas is delivered into the airway simultaneously in forward and reverse directions. Of approaches evaluated to date, r-TGI and bi-TGI appear to be the most effective in preventing increased total PEEP during TGI administration.[14,16–18] Delgado et al postulate that simultaneous insufflation of gas in opposite directions might reduce or eliminate the back pressure that produces auto-PEEP.[30] Four catheter configurations were studied in an artificial lung model during PCV under constant minute ventilation conditions (Fig. 4). During bi-TGI and r-TGI, levels of total PEEP were lower during PCV than during c-TGI and e-TGI, at each of the 3 inspiratory-expiratory ratios studied (1:1, 1:2, 2:1), and CO_2 elimination efficiency was comparable. If supported in animal and human studies, these findings suggest an important advantage of r-TGI and bi-TGI over c-TGI and e-TGI delivered in a forward flow direction.

Monitoring auto-PEEP can be quite challenging. The end-expiratory occlusion method cannot be used during any TGI technique that continues to deliver gas into the lungs, because a static end-expiratory pressure cannot be achieved.[26] The use of respiratory inductive plethysmography also presents difficulties because substantial baseline drift occurs over time.[9,20] In addition, respiratory inductive plethysmography is not widely available. We have found that a 5 cm H_2O reduction in extrinsic (ventilator set) PEEP is typically required to keep total PEEP constant during c-TGI at 10 L/min.

Carbon Dioxide Elimination Efficiency

The efficiency of TGI is best measured by monitoring arterial carbon dioxide tension (P_{aCO_2}). However, this measurement is invasive and episodic and there are few data to provide guidelines about optimal times to measure P_{aCO_2} following initiation of TGI. Hoffman et al[19] reported no difference in P_{aCO_2} measured at 30 minutes and 60 minutes after initiation of TGI in 8 acute respiratory distress syndrome patients. They therefore concluded that effectiveness of TGI was evident within 30 minutes, suggesting that the efficacy of this intervention could be rapidly determined.

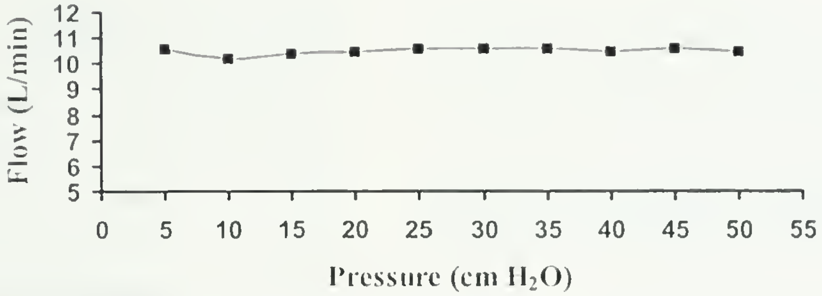

Fig. 2. Insertion of a prototype flow relief valve into the ventilator circuit maintains consistent venting of 10 L/min gas flow and provides consistent delivered volume during continuous tracheal gas insufflation under varying inspiratory pressures.

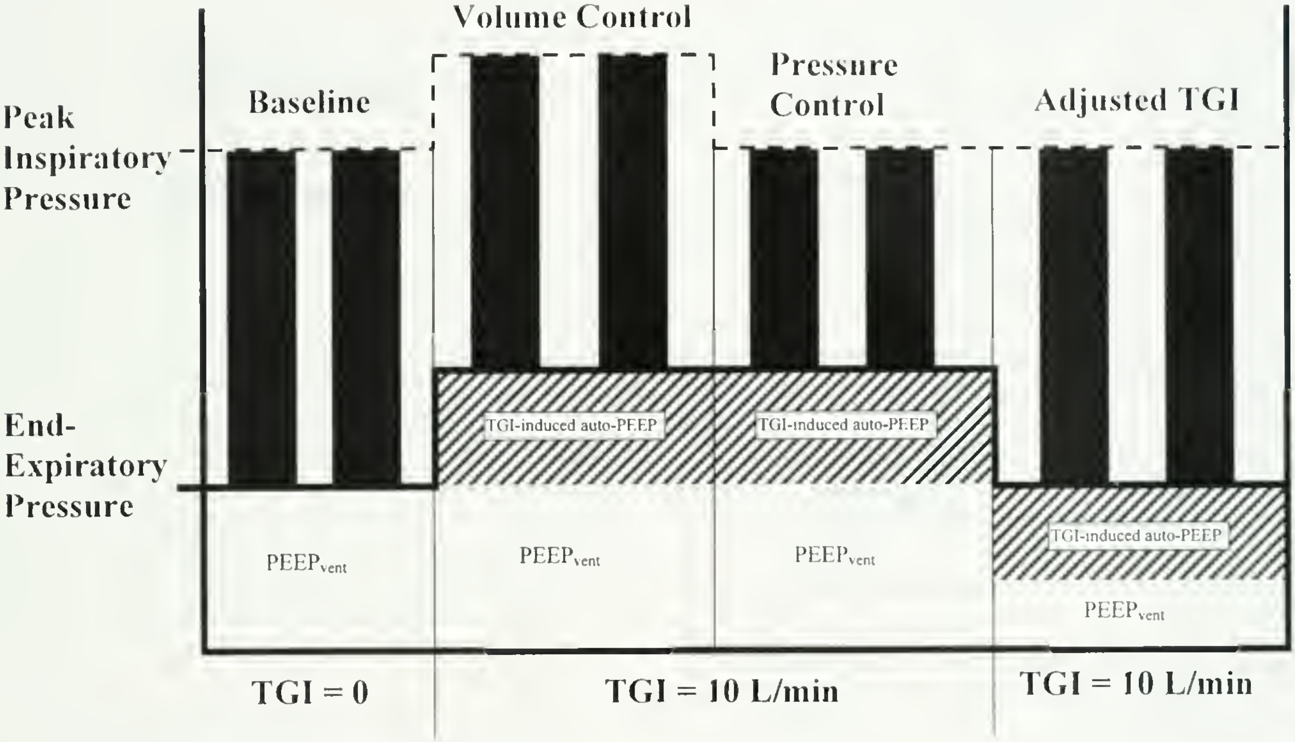

Fig. 3. Theoretical framework that illustrates interactions between tracheal gas insufflation (TGI), ventilator mode, minute ventilation (tidal volume), and intrinsic positive end-expiratory pressure (auto-PEEP). During volume-controlled ventilation, when ventilator PEEP (height of clear areas labeled PEEP_{vent}) is left constant, tidal volume (height of black bars) remains constant, but there is an increase in end-expiratory pressure and, therefore, peak airway pressure, because of TGI-induced auto-PEEP (height of cross-hatched areas). During pressure-controlled ventilation, when PEEP_{vent} and peak airway pressure are kept the same as baseline, tidal volume excursions (and, thus, minute ventilation) are reduced because of TGI-induced auto-PEEP. When PEEP_{vent} is reduced by an amount equivalent to TGI-induced auto-PEEP, tidal volume, peak airway pressure, and total PEEP remain the same as baseline during pressure-control ventilation. (Adapted From Reference 20, with permission.)

Two studies evaluated the use of monitoring end-tidal carbon dioxide tension (P_{ETCO_2}), obtained by capnography, as a semiquantitative indicator of TGI efficiency.[9,12] In 8 patients with acute respiratory failure, Ravenscraft et al[9] found a moderate (r = 0.68) correlation between the percentage reduction in P_{aCO_2} ($\%\Delta P_{aCO_2}$) as a function of the percentage reduction in P_{ETCO_2} ($\%\Delta P_{ETCO_2}$) from the baseline value (Fig. 5). Kuo et al[12] compared the $\%\Delta P_{aCO_2}$ and $\%\Delta P_{ETCO_2}$ from baseline values in 20 adults with acute respiratory distress syndrome. The $\%\Delta P_{ETCO_2}$ correlated significantly ($r = 0.75$; $p < 0.001$) with the $\%\Delta P_{aCO_2}$ (Fig. 6).[7] Both authors concluded that, although P_{ETCO_2} is a poor estimate of P_{aCO_2} in patients with respiratory failure, these data may justify use of P_{ETCO_2} as a monitor of trends regarding changes in CO_2 elimination efficiency during TGI.

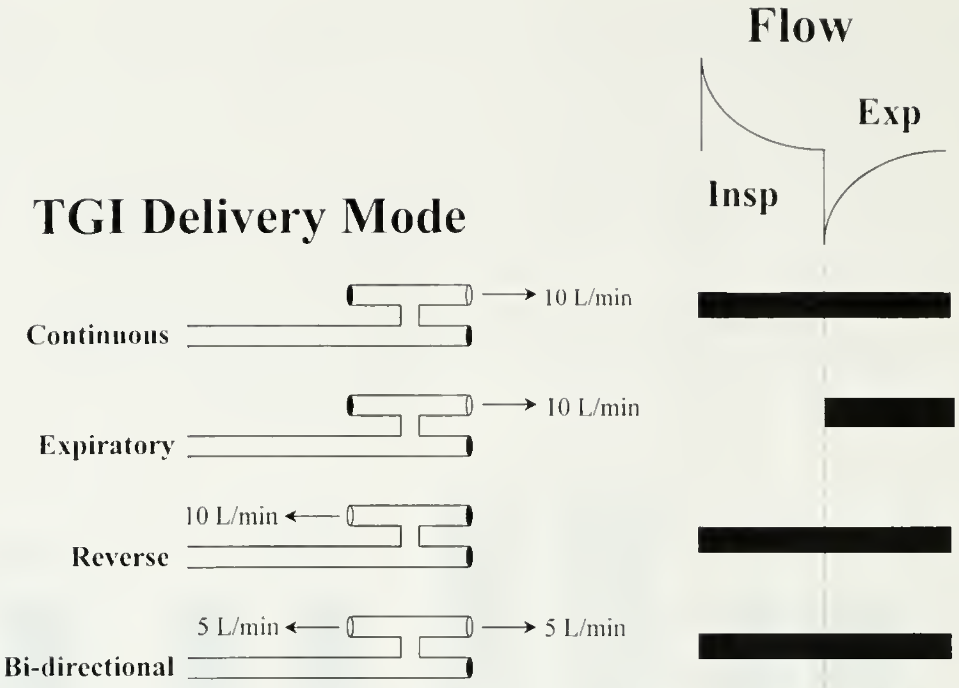

Fig. 4. Prototype H-shaped-tip catheter configurations and flow patterns with different tracheal gas delivery modes.

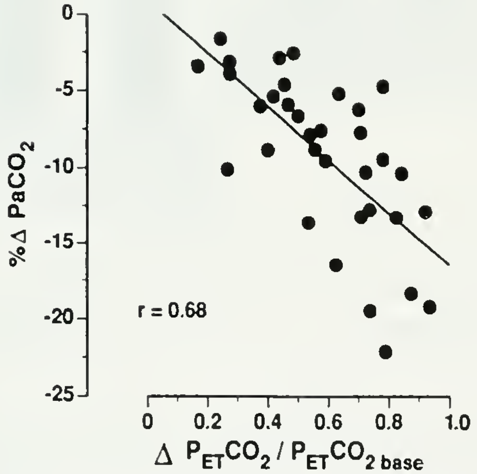

Fig. 5. Percentage reduction of arterial carbon dioxide tension ($\%\Delta P_{aCO_2}$) from baseline as a function of the reduction in the end-tidal carbon dioxide tension (P_{ETCO_2}) from the baseline value ($P_{ETCO_{2base}}$) in 8 patients suffering acute respiratory failure and receiving tracheal gas insufflation. As the difference between P_{ETCO_2} and $P_{ETCO_{2base}}$ increased, P_{aCO_2} decreased. (From Reference 9, with permission.)

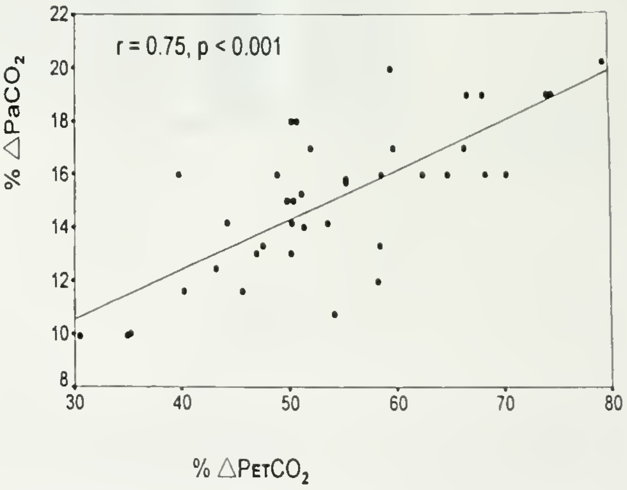

Fig. 6. Relationship between percent reduction of arterial carbon dioxide tension ($\%\Delta P_{aCO_2}$) and percent reduction in end-tidal carbon dioxide tension ($\%\Delta P_{ETCO_2}$) from baseline values in 20 patients suffering from acute respiratory distress syndrome and receiving tracheal gas insufflation. (From Reference 12, with permission.)

Humidification

Humidification during TGI is an important issue that has received little study. The data available are mainly derived from studies investigating long-term use of transtracheal oxygen delivery in spontaneously breathing patients, whereas most TGI studies have been conducted in patients on full ventilatory support.

The respiratory tract performs a role in a variety of functions, including ventilation, gas conditioning, production and inactivation of bioactive substances, filtering, smell, communication, mucociliary transport, and surfactant production.[27,28] When a patient is endotracheally intubated, some of these functions are interrupted or bypassed, whereas others (including the integrity of secretions) are affected by the temperature and humidity of the inspired gas. In a preliminary report, Shapiro et al[29] found that TGI significantly cooled the central airways and that the conditioned gas delivered by the ventilator cannot compensate for this effect. Further, patients who require mechanical ventilation are more likely than spontaneously breathing patients to be more susceptible to having more secretions and more susceptible to the effects of jets of gas striking the tracheal wall.[25]

In airway mucosa there are basically 3 heat and moisture paths. The heat and moisture given off during inspiration, the recovered heat and moisture during exhalation, and the heat and moisture obtained from systemic reserves.[31] Therefore, it is logical to assume that the airways may be exposed to adverse effects if the inspiratory gas delivered by the ventilator and TGI system is not conditioned. Fletcher et al[32] reported the development of an endotracheal mass (mucus plug 1.8 × 2.5 cm) after 9 days of transtracheal oxygen delivery at 3 L/min. In a report by Burton et al[33] a 50-year-old man with chronic obstructive pulmonary disease and pulmonary fibrosis developed severe dyspnea and subsequently expired after approximately 15 days of transtracheal oxygen delivery with a flow of 3 L/min. The autopsy report revealed a mucus plug obstructing the patient's trachea. Hoffman et al[3] studied 40 patients receiving long-term transtracheal oxygen delivery and reported formation of mucus balls on the tip of the catheter in 25% during the time the tract is immature and the catheter is cleaned in place. A retrospective study of 56 patients using transtracheal oxygen catheters from 2 days to more than 6 years found a mucus plug prevalence rate of 38%.[34]

Most tracheal gas insufflation studies completed on humans and animals have been short-term and either do not report whether the TGI gas was heated and humidified or report that a nonheated, dry gas was used. When a heated and humidified system was used to condition the gas during intratracheal pulmonary ventilation, Kolobow et al[16] reported no damage to the tracheal mucosa and no encrustation. Danan et al[15] reported using humidified and warmed TGI in 9 premature newborns for up to 31 days (mean 17 d). The tracheas of 3 newborns who died were examined and found normal. No problems due to mucus plug formation were reported in any subjects. In that study, the humidified and warmed (Fisher and Paykel MR600, Auckland, New Zealand) gas was derived from the inspiratory line of a conventional ventilator circuit and forced into the TGI capillaries of an endotracheal tube using a membrane pump (110 cm H_2O) at a rate of 0.5 L/min. Kuo et al[12] reported using humidified, nonheated TGI gas with a straight-tip catheter continuously for up to 72 hours in 12 patients. TGI was discontinued in 2 patients because of intratracheal catheter obstruction, which occurred after 58 and 67 hours, respectively. Both events were detected immediately (by the whistling sounds emitting from the pressure release valve on the humidifiers) and caused no problems. When removed, both catheter tips were obstructed by inspissated mucus plugs. Nine patients completed up to 72 hours of TGI. Eight had no change in the tracheobronchial mucosa, and one had focal mucosal erythema adjacent to the carina on bronchoscopic examination.

To date, there have been no reports of airway occlusion secondary to mucus plugging during TGI. Nevertheless, it is important to monitor for this complication and institute preventive measures. If the expiratory circuit of the ventilator becomes occluded, the TGI catheter could deliver a large volume of gas, potentially resulting in serious barotrauma and hemodynamic compromise.[9] A reliable mechanism to detect an increase in pressure and stop TGI flow needs to be developed to enable safe long-term provision of TGI.[9,25,26] Until such mechanisms are commercially available, using prophylactic measures to avoid problems is important. These include monitoring for increases in PIP due to possible mucus plugging and removal of the TGI catheter every 8–12 hours to assess for plugs. One difference between the gas conditioning techniques described by Kolobow et al[16] and Danan et al[15] and that described by Kuo et al[12] was that the latter study did not heat the inspired gas. This observation suggests that additional attention needs to be given to developing a mechanism to heat and humidify TGI gas delivered in all systems.

Summary

The use of TGI as an adjunct to mechanical ventilation in the intensive care unit environment requires conventional monitoring, regardless of the TGI system used. In addition, monitoring of changes in V_T, PIP, and P_{aCO_2} are necessary. Monitoring for the development of auto-PEEP is also indicated. Optimally, gas insufflated by the TGI catheter should also be conditioned by addition of heat and humidity to prevent mucus plug formation and damage to the tracheal mucosa. Finally, patients should be monitored for increases in PIP due to mucus plugging, and the TGI catheter should be removed every 8–12 hours to assess for plugs.

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Intratracheal Catheters As Drug Delivery Systems

Neil R MacIntyre MD FAARC

Introduction
Drugs That Have Benefit If Delivered through the Airways
Aerosol Versus Instillation Techniques for Administering Drugs into the Lungs
Intratracheal Aerosol Delivery
Combining an Intratracheal Aerosol System with Tracheal Gas Insufflation
Summary

Medication delivery into the lungs can be used to provide a high therapeutic index for agents targeted to specific lung diseases. In addition, the lung can be used as a portal of entry for other agents targeted to systemic diseases. Delivery of medications into the lung can be accomplished by either instillation or aerosolization. Instillation approaches are limited by the fluid volume that can be given safely, and instilled liquids distribute according to gravity. In contrast, aerosolization approaches can deliver larger volumes over longer periods and aerosols distribute according to ventilation. In the mechanically ventilated patient, externally generated aerosols have very poor lung delivery because the endotracheal tube functions as a barrier to aerosol passage. Novel aerosol generating systems at the ends of small-diameter catheters that can be placed into the trachea (or beyond) are being developed to address this. In vitro testing has shown these systems to be capable of producing appropriately sized particles, with high rates of lung deposition. These catheters could be coupled with tracheal gas insufflation systems, not only to deliver therapeutic aerosols but also to create water aerosols to supply necessary humidification during tracheal gas insufflation.

Key words: tracheal gas insufflation, catheters, pulmonary drug delivery, aerosols, instillation. [Respir Care 2001;46(2):193–197]

Introduction

Delivery of drugs into the lungs through airways is done for two purposes.[1-3] First, a high therapeutic index (efficacy/toxicity) can be achieved for a variety of lung conditions when the drug is delivered directly into the lung, as compared to through the systemic circulation. Examples include β agonist bronchodilators and steroids to treat airway inflammation. Second, in recent years there has also been considerable interest in using the lung as a means to access the systemic circulation. The lung is thus used as an entry site rather than a therapeutic target for a drug.[4]

Drugs That Have Benefit If Delivered Through the Airways

At the present time, the United States Food and Drug Administration has approved several classes of drugs for use as aerosols or as instilled drugs to treat lung disease. These include the bronchodilators, corticosteroids, mucolytics, and surfactants. There are also published reports of a large number of other Food and Drug Administration-approved drugs that have been tried as aerosols or instil-

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Table 1. Examples of Drugs with Demonstrated Pulmonary Delivery Efficacy

Antibiotics
Insulin
Heparin
Prostacyclin
Alpha-1-antitrypsin
Leukotriene modifiers
Indomethacin
Surfactants
Interferons
Hormones
Colony stimulating factors/erythropoietin
Somatostatin

Note: These are examples of drugs approved by the United States Food and Drug Administration for human use but not specifically approved for delivery via aerosol or airway instillation and for which there is evidence of efficacy with pulmonary delivery.

lations (Table 1).[5] These drugs, however, are not officially approved for direct instillation or aerosolization to the lung. Note that of the drugs designed to treat lung diseases, aerosolized antibiotics are the most widely studied.[6] There are also emerging data on the use of insulin administration through the lung as a way to treat the systemic disease diabetes mellitus. Prostacyclin is another drug used to treat systemic disease (pulmonary and systemic vascular diseases) that is being administered through the aerosol route. Finally, there is a wide array of experimental therapeutics being investigated as possible aerosols.[5] As noted above, these drugs are either aimed at treating specific lung injuries/abnormalities or else are administered to the lung in order to access the systemic circulation. Table 2 lists many of those drugs.

Aerosol Versus Instillation Techniques for Administering Drugs into the Lungs

There are two conceptually different ways of delivering a medication into the lungs. The first is to insert a delivery catheter either through the larynx or through an artificial airway (eg, endotracheal or tracheostomy tube) and directly deliver a bolus of drug. The second way is to generate an aerosol external to the patient and have either the patient's spontaneous ventilatory efforts or a positive pressure breath from a ventilator deliver the aerosol into the lung.

Table 2. Examples of Experimental Drugs Administered via Aerosol

Antibodies
Gene transfer vectors
Antioxidants
Antiproteases
Cytokine modifiers
Arachidonic acid metabolite modifiers
Vaccines

Advantages to the instillation technique are that large volumes can be delivered quickly. There is a limit, however, to how much fluid can be delivered over a short period of time (eg, lung mechanics and gas exchange begin to alter with instillations of as little as 1 mL/kg).[7] Moreover, there is often a transient "bolus" effect on both gas exchange and hemodynamics, especially if the bolus volume is substantial.[8] The distribution of an instilled medication is usually gravity-dependent. Mechanically ventilated patients thus generally have the distribution going into the posterior regions of the lung, whereas an upright patient breathing spontaneously would have an instilled bolus go caudally.[9] Surfactant is the most common instilled drug, and it is a common technique to rotate the patient through various axes to assure optimal distribution. Experimental delivery of perflubron also utilizes a similar approach.

The aerosol route offers the opportunity to deliver medications at a more constant rate over a much longer period of time. Aerosolized medications tend to distribute according to ventilation, as opposed to gravity.[9,10] Other factors affecting delivery of an aerosol include particle size and velocity, because these affect deposition through impaction, sedimentation, and diffusion.[10] In general, the higher the flow rate and the larger the particle size, the more likely the aerosol is to be deposited in the major airways. In contrast, smaller particles moving at a slower velocity tend to penetrate deeper into the lung and deposit in more distal lung regions. Conceptually, drug targeting to specific regions of the lung could be accomplished by manipulating these aerosol characteristics.

Table 3. Effect of Flow Rate and Endotracheal Tube Diameter on the Deposition of an Externally Generated Aerosol

Air Flow (L/min)	ETT (mm)	Percent Deposition*	
		Filter	ETT
30†	3	2.5	45.7
	5	10.7	43.7
	6	12.3	40.1
20‡	6	4.0	52.0
	9	10.0	30.0
60‡	6	5.0	57.0
	9	11.0	40.0

ET = endotracheal tube

*Percent of nominal dose (by weight) of metaproterenol or albuterol administered with a swivel adapter connected to the ETT. Metered-dose inhaler actuated into a continuous flow of air

†Data from Crogan SJ, Bishop MJ. Delivery efficiency of metered dose aerosols given via endotracheal tubes. Anesthesiology 1989;70(6):1008–1010

‡Data from Taylor RH, Lerman J. High-efficiency delivery of salbutamol with a metered-dose inhaler in narrow tracheal tubes and catheters. Anesthesiology 1991;74(2):360–363.

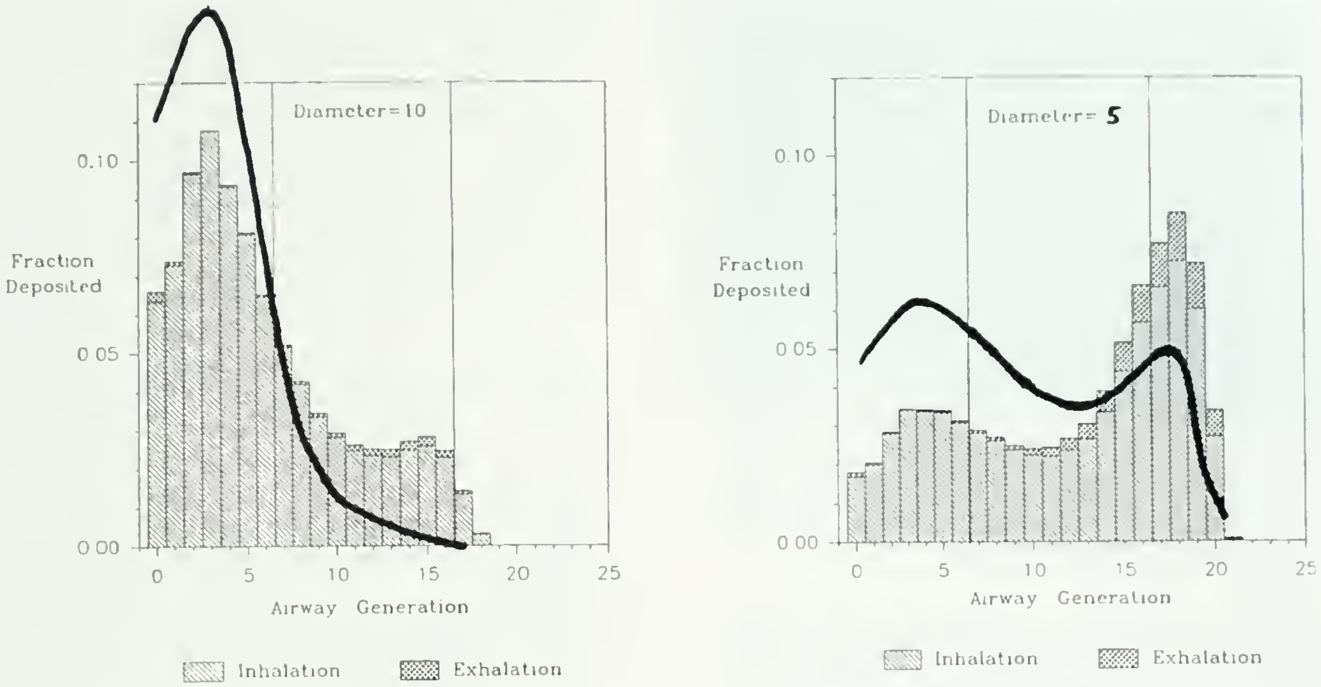

Fig. 1. Predicted distribution of aerosol particles of different sizes when the aerosol is generated within the trachea. Data were generated from a computer model of the Weibel lung, using gas flow of 0.5 L/s. The shaded bars indicate predicted delivery using intratracheal generation of 10 μm particles (left panel) and 5 μm particles (right panel) and the solid lines are the expected distribution from an externally generated aerosol. Note that when the aerosol is generated in the airway, larger particle sizes penetrate deeper into the lung.

The presence of an endotracheal tube (ETT) poses a particular problem with the delivery of a therapeutic aerosol from an external generator.[11–13] In adults these tubes are generally 7–9 mm internal diameter, up to 30 cm in length, and are often bent up to 90°. Because of these features, the ETT is an important site of impaction of externally generated aerosol particles. Indeed, therapeutic aerosol through an ETT can be as little as 20% of the delivery that would normally occur through the natural upper airway (Table 3). Table 3 illustrates the combined effects of increasing air flow velocity and reducing ETT diameter on deposition of an externally generated aerosol within the tube. The ETT material may also impact deposition. Specifically, certain types of plastics may promote aerosol rain-out and the presence of an electrostatic charge on the tube can also reduce the ability of the aerosol to pass through it.[12,13]

Intratracheal Aerosol Delivery

Because of the impact of the ETT, attempts have been made in recent years to develop strategies to generate an aerosol beyond the ETT (ie, within the trachea or bronchi). Generating the aerosol distal to the tube offers not only the benefit of larger amounts of aerosol reaching the lung, but it can also affect the optimal particle size distribution needed for effective delivery. Specifically, if the aerosol could be generated distal to the upper airways and ETT, larger particle sizes conceivably could be utilized.[14] To illustrate this concept, Figure 1 shows predicted deposition of aerosols of different particle size using a computer lung model and a 0.5 L/s inspiratory flow when the aerosol is generated in the trachea, as opposed to externally.[14] Note from Figure 1 that larger particle sizes generated intratracheally would be expected to penetrate deeper into the lung and have a better therapeutic effect than the same size particles generated externally. Another obvious effect of an intra-airway aerosol generator is that the device could be guided either radiologically or under direct vision to specific lung regions for very directed drug targeting (eg, radiopharmaceuticals for cancers, antibiotics for pneumonia).

The first attempt to produce an aerosol within the lung was the simple use of a long extender on a standard metered-dose inhaler.[15] Though this approach can place the aerosol exit site inside the airway, the very high velocities that come out of a metered-dose inhaler causes substantial impaction of the aerosol at the carina and in the mainstem bronchi. Because of this, this technique has not been used extensively. Another limitation is that only drugs packaged as metered-dose inhalers could be delivered in this fashion.

In 1994 a novel system was introduced that uses a small diameter (1 mm) multi-lumen catheter capable of producing an aerosol with a therapeutically appropriate particle

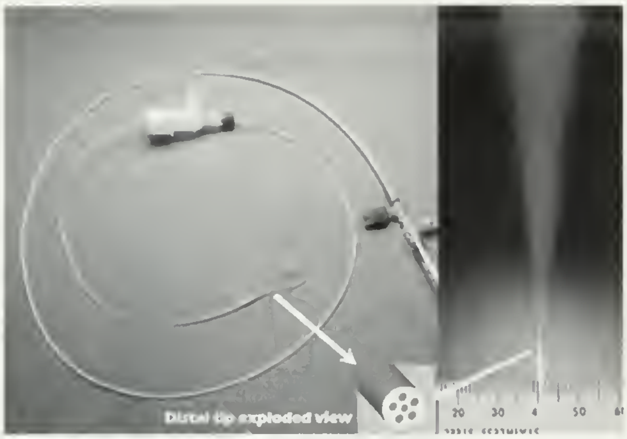

Fig. 2. Schematic of a prototype aerosol-generating catheter. Liquid is delivered through the central lumen and gas is delivered through the peripheral lumens. At the interface of these lumens, at the catheter tip, an aerosol is created. (Photo courtesy of Trudell Medical International, London, Ontario, Canada.)

Table 4. Regional Deposition in an Intubated, Mechanically Ventilated Glass Lung Model of an Aerosol Generated External to the Patient, As Compared to an Aerosol Generated within the Trachea

	Percent Deposition In			
	Circuit	ET tube	Airways	Alveoli
Externally-generated	79	10	4	5
Catheter	18	15	56	11

size.[16] Figure 2 shows a schematic of this catheter system, in which the liquid is delivered down the central lumen and gas is delivered through the peripheral lumens. The tip of this catheter is tapered such that at the interface of the liquid and gas lumens an aerosol can be created. Depending on gas flow (typically 0.8–1 L/min), 0.5–2.0 mL/min of liquid can be aerosolized, with particle sizes in the therapeutic range. In a glass model of the first 4 branches of the human tracheobronchial tree this device has been shown to increase alveolar and airway deposition, compared to a standard external nebulizer aerosol delivered through an endotracheal tube (Table 4).

There are other conceptual approaches to placing the aerosol generator within the lung. For instance, a miniaturized ultrasonic system might be placed at the tip of a catheter, over which a liquid is delivered. One of the advantages of this approach over the jet catheter described above is that no extra flows or pressures are delivered to the lung and the particle velocity is virtually zero. Two other recently introduced aerosol generating techniques might also be miniaturized to produce a high-quality, low-velocity aerosol within the airway.[17] The first is a device that uses a high-velocity oscillating membrane to create aerosols by forcing liquids through tiny pores. The second approach places an electric charge on an extruded liquid to break the surface forces and create an aerosol. In the future it is also conceivable that dry powder generators might also be miniaturized and placed at the distal end of a catheter inserted through the ETT.

Combining an Intratracheal Aerosol System with Tracheal Gas Insufflation

Tracheal gas insufflation (TGI) requires a small-diameter catheter to be placed within the ETT, with its distal tip proximal to the carina. The primary purpose of TGI is to supply fresh gas, principally through the expiratory phase, to clear effective dead space.[18] The conceptual advantage is that smaller tidal volume can be used and thus lower end-inspiratory volumes and pressures can be applied to the chest during mechanical ventilation. This may be particularly important when conventional mechanical ventilatory strategies are producing excessive end-inspiratory "stretch" and efforts are being made to reduce this.

Because TGI has a catheter already within the ETT, coupling it to an intratracheal aerosol-generating system is certainly possible. For the jet catheter design noted above, the TGI gas flow could actually double as the gas being used to generate the aerosol during inspiration. Other systems that do not require gas flow could have the fluid catheter attached to the TGI catheter and the aerosol generator mounted on the distal end.

Another potential use of an intratracheal aerosol generator during TGI is to condition the TGI gas flow with moisture. On current TGI systems under development, the humidification of gases generally takes place before gas enters the TGI catheter, often using separate humidification systems that are not designed for the high pressures required for TGI. The intratracheal techniques described above, however, could be used to generate a bland aerosol to provide the necessary airway moisture and thereby obviate a separate humidification system.

Bland aerosol generation has been described previously as a technique for conditioning dry gas during mechanical ventilation.[19] Its safety and efficacy, however, are controversial. On the one hand, several studies have shown that small-particle bland aerosols from standard nebulizers can significantly impair gas exchange in as little as 30 minutes.[20–22] The mechanism for this is thought to be occlusion of small airways by 1–5 μm water droplets. It should be noted, however, that all of those studies used systems that generated several-fold more water than would be required for normal humidification (40 mg or microliters of water per liter of ventilation). Moreover, the particle sizes, though appropriate for drug delivery to the lung, were excessively small for targeting moisture delivery to the major airways. In contrast, a bland aerosol TGI gas-conditioning strategy would use much smaller liquid volumes

(TGI gas flow is only a fraction of total minute ventilation), would generate fewer very-small particles, and would be delivered only during exhalation. The small amount of aerosolized water with this approach would thus be delivered primarily to the trachea and endotracheal tube where atelectasis concerns are not an issue and where the water particles can also serve as an evaporative source of moisture. It therefore may be quite reasonable to use bland aerosol generation of appropriate volumes and particle size to provide the airway moisture necessary for effective TGI. Supporting this concept is the safe and effective use of a jet nebulizing system to provide moisture to jetted gas at the proximal ETT during high-frequency jet ventilation.[23]

Summary

A growing number of medications are being developed that are designed to be delivered into the lungs. Aerosol delivery of these medications from an external generating system into the patient's lungs, even under the best of circumstances, is less than 50%, and delivery efficiency usually drops to less than 10% in the presence of an ETT. Novel aerosol generating systems at the end of small-diameter catheters that can be placed into the trachea (or beyond) are being developed to address this. In vitro testing has shown these systems to be capable of producing appropriately sized particles, with high rates of lung deposition. These catheters could be coupled with TGI systems not only to deliver therapeutic aerosols but also to create water aerosols to supply the necessary moisture for TGI gas flow.

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Tracheal Gas Insufflation: Overcoming Obstacles to Clinical Implementation

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Introduction

Tracheal gas insufflation (TGI) is the injection of gas into the distal trachea to functionally reduce dead space and thereby improve the efficiency of mechanical ventilation. In the context of a lung-protective mechanical ventilation strategy, the objective of TGI is to permit a reduction in tidal volume and thus alveolar distending pressure. In other applications, TGI may be useful to decrease the ventilation requirement for a target arterial partial pressure of carbon dioxide. At this conference, clinical investigators involved in the development of TGI reviewed both technical aspects and data from TGI experiments in lung models, animals, and human subjects. Their written commentaries comprise the papers published in this issue of RESPIRATORY CARE.

Despite considerable academic and clinical interest, approved TGI systems are not available in North America and use is thus limited to clinical studies. Commercial systems to provide TGI are being developed and several may become available in the near future. The use of jury-rigged systems to provide TGI is discouraged, primarily because of the potential for complications related to the TGI-ventilator interaction. When TGI systems become available, the information published in this issue of RESPIRATORY CARE should provide valuable information for clinicians using this technique.

Because it was attended by clinicians, researchers, and industry representatives, this conference provided a unique opportunity to discuss a variety of issues related to implementation of TGI. Clinicians desire a system that is safe, efficacious, easy to use, and affordable. Industry, in trying to address these concerns, is faced with regulatory issues that that must be overcome before TGI systems can marketed.

At the end of this conference a series of questions were presented to the attendees as points for discussion. The group responses to these questions provide the basis for this summary.

What Additional Lung Model and Animal Studies Are Needed?

Many lung model and animal model studies of TGI have been conducted and much has been learned from these. Some additional studies of this type may be necessary to further our understanding of the mechanisms whereby TGI facilitates CO_2 clearance. For example, in various clinical situations (eg, airway obstruction, unilateral lung injury, parenchymal disease) what TGI flows are needed and when should they be applied during the respiratory cycle? Another important role of lung model studies is the characterization of commercial systems before they are used clinically. Prototypes can be tested on models to understand their functional characteristics and to identify issues that might have clinical relevance. This relates to both TGI flow controllers and the catheters or endotracheal tubes used for TGI. For example, animal models may be necessary to assure that airway mucosal injury does not occur. Animal and/or lung model studies are also necessary to demonstrate adequate humidity delivery with these devices.

What Clinical Studies Are Needed?

The consensus of the group was that the beneficial effects of TGI on lung distention should improve clinical outcomes. However, it was recognized that studies using mortality or ventilator-free-day end points would require large clinical trials that are impractical. Given the perceived safety of properly designed TGI systems, the group therefore agreed that physiologic studies of TGI are all that is currently needed to demonstrate safety and efficacy. Appropriate physiologic outcomes for TGI include reduction in arterial partial pressure of carbon dioxide, reduction

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in tidal volume and peak alveolar pressure, and reduction in minute ventilation.

What Clinical Study Design Is Necessary?

For assessment of physiologic outcome variables, a crossover design should be adequate. Although a randomized design can be used in which patients are assigned to receive TGI or standard therapy without TGI, this approach adds little scientific value over a crossover design and is considerably more costly to perform. Moreover, the physiologic effects of TGI in crossover studies can be assessed in several hours. Longer studies (ie, days) of TGI are necessary to assess long-term effects of TGI—such as safety, stability of physiologic benefit, and device reliability in the clinical arena. The effects of TGI should be assessed for patients who are fully supported by the ventilator as well as for those who are triggering the ventilator.

What Safety Variables Should Be Assessed?

Safety issues with TGI that should be studied include humidification, intrinsic positive end-expiratory pressure, airway injury, triggering, device reliability, and TGI-ventilator interactions. Note that many of these safety issues do not require human studies to answer the important questions. However, clinical verification of these issues in human observational studies should be done.

In What Patient Populations Should Tracheal Gas Insufflation Be Studied?

Because of concern about over-distention lung injury for acute respiratory distress syndrome patients, it is reasonable to study the use of TGI in these patients first. However, overdistention lung injury can also occur in other lung diseases that require mechanical ventilation. For instance, TGI may have important applications for patients with obstructive lung disease in which regional overinflation is common. Indeed, any lung disease requiring mechanical ventilation and in which regional overdistention is possible could benefit from TGI.

Design Issues: Should Tracheal Gas Insufflation Be Applied with a Catheter or Special Endotracheal Tube? What Is the Best Design? Should It Be Applied Continuously or Intermittently?

TGI can be applied using either a catheter passed through the lumen of the artificial airway or through a lumen designed into the wall of the artificial airway. Neither approach is clearly superior to the other. However, application of TGI should *not* require reintubation of the patient.

The importance of catheter tip design (ie, forward flow, reverse flow, or combination) is unclear. Forward flow seems to augment gas mixing, but reverse flow seems to reduce the potential for increasing intrinsic positive end-expiratory pressure. Both designs have been shown to be effective, and there are currently no data that show a clear superiority of one design over the other.

The choice of continuous versus expiratory-phase TGI is often based on the bias of the investigator. Continuous TGI uses a simpler gas delivery design. With continuous TGI, however, pressure or flow relief valves are needed to account for the effect of the TGI flow during the inspiratory phase (ie, device design must make TGI flow invisible during the inspiratory phase).

What Needs To Be Monitored and/or Alarmed during Tracheal Gas Insufflation?

Although exhaled CO_2 analysis can be used to guide TGI settings, TGI systems should not require the routine use of capnography. The system should allow the ability to monitor intrinsic positive end-expiratory pressure or dynamic hyperinflation secondary to TGI. It is helpful to know the TGI volume insufflated. A mechanism should be in place so that the volume measured by the ventilator can be corrected for the TGI flow. A mechanism must be provided to detect an obstruction in the artificial airway.

What Cost Barriers Are Important to the Introduction of Tracheal Gas Insufflation?

There are costs associated with the TGI controller, the catheter, and the education of clinicians to use the system. Additional costs include those required to do the necessary studies to demonstrate safety and efficacy to clinicians, regulatory agencies, and payers. The cost of the system should be commensurate with the benefit derived from TGI.

Summary

This commentary represents our review of the group response to questions regarding the obstacles to clinical use of TGI. This, along with the accompanying papers in this issue of RESPIRATORY CARE, should help industry, regulators, investigators, and clinicians with the introduction of this technology to the care of mechanically ventilated patients.

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Reviews of Books and Other Media. Note to publishers: Send review copies of books, films, tapes, and software to RESPIRATORY CARE, 600 Ninth Avenue, Suite 702, Seattle WA 98104

Interventional Pulmonology. John F Beamis Jr MD and Praveen N Mathur MD, editors. New York: McGraw-Hill. 1999. Hardcover, illustrated, 332 pages, $145.

The authors, in the preface to **Interventional Pulmonology**, define the book as a reference source for interventional pulmonologists as well as for pulmonologists referring or caring for patients who have undergone the procedures described in this book. While many of the chapters deal with procedures that are well within the purview of most practicing pulmonologists, such as thoracentesis, closed pleural biopsy, transbronchial needle aspiration, and chest tube placement, others are more likely to be performed in referral centers. In fact, in the preface the authors support the definition of interventional pulmonology as those procedures that require training beyond that of the typical pulmonary and critical care fellowship. The primary audience for this book is practicing pulmonologists. However, respiratory therapists who assist in these procedures would also find this text useful.

The book is organized into three sections. The first part describes various aspects of therapeutic bronchoscopy; the second part deals with medical thoracoscopy; and the final part groups miscellaneous procedures such as percutaneous tracheostomy, transbronchial needle aspirations, and thoracentesis.

The initial chapters in the section on bronchoscopy include a general overview of bronchoscopy and maintenance of the flexible bronchoscope, which, while of interest to respiratory therapists, are adequately addressed in other sources.

The chapter on laser bronchoscopy, while thorough, includes a fair amount of laser physics, which may be of limited use to the readers of RESPIRATORY CARE, but the section dealing with contraindications and indications for laser bronchoscopy, is useful from the standpoint of referring pulmonologists. The section on stents, including separate chapters for both silicone stents and expandable wall stents, is particularly well done. The illustrations are very helpful. The discussion is quite thorough regarding the pros and cons of various stents. It includes a very thorough review of the silicone stents currently available.

The second section of the book is devoted to medical thoracoscopy. This section addresses routine chest tube placement, but the bulk of the section deals with thoracoscopic inspection and biopsy of the pleural space, and management of pleural space problems such as pneumothorax and pleural malignancy. The author's definition of medical thoracoscopy emphasizes the fact that this procedure is performed by pulmonologists, as opposed to thoracic surgeons, and that the primary role is diagnosis rather than treatment. In this reviewer's opinion, these procedures are more commonly within the realm of thoracic surgery in most communities. Nonetheless, the description and illustrations are quite informative and the chapters are well written. They include a very nice, abbreviated review of the literature regarding pleurodesis with talc versus other agents.

The final section of the book contains chapters dealing with various procedures. These include transbronchial needle aspiration, transtracheal oxygen therapy, thoracentesis, enclosed pleural biopsy, and percutaneous tracheostomy. Many of these are within the traditional training of most pulmonologists, and may prove valuable to respiratory therapists.

In general, the book is written from a very practical standpoint. This is a text about procedures, and the authors certainly have kept that focus in mind. The chapters are relatively brief and reasonably comprehensive. At the end of most chapters there is a section (written by an expert) entitled "How I Do It," with a step-by-step description of his or her technique. The book, of course, is not a substitute for actual training in these techniques by an expert, but would serve as a useful reference to pulmonary physicians performing these procedures, both from a standpoint of having a ready reference regarding an expert's technique and to provide a succinct review of the procedure and the relevant literature associated with it. The book does have a place in a respiratory therapy department library, particularly in facilities that are involved with performing any of the invasive procedures discussed in the text.

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Mosby's Diagnostic and Laboratory Test Reference, 4th edition. Kathleen Deska Pagana PhD RN and Timothy James Pagana MD. St Louis: Mosby. 1999. Hardcover, 967 pages, $28.95.

This diagnostic and laboratory test reference is well designed. The handbook uses a consistent format that allows tests and diagnostic procedures to be found quickly without loss of the discussion needed for a complete understanding. The following information is provided:

Name of the test: Complete name with a list of abbreviations and alternative test names following each main entry.

Type of test: This section helps the reader identify the source of the laboratory specimen (eg, blood) or type of diagnostic procedure (eg, chest radiograph).

Normal findings: Normal values are listed for infants, children, adults, and the elderly. Where appropriate, separate values are listed for male and female.

Possible critical values: These are threshold values that when crossed generally require immediate intervention.

Test explanation and related physiology: This section includes information about the test, specific indications for the test, how the test is performed, what disease pathology or disorder the results may show, and related pathophysiology.

Contraindications: These data alert the reader to patients who should not have the test.

Potential complications: This section describes potential problems that may require astute assessments and interventions.

Interfering factors: Many factors such as drugs can interfere with test results (a special icon is used to alert the user when drugs might interfere with the test).

Procedure and patient care: This section is divided into before, during, and after time sequences. The procedure for conducting the test is described and the information that should be presented to the patient is discussed for each time interval.

Home care responsibilities: Early discharge from the hospital and outpatient testing were the motivating forces for including this section. The authors believe "that patients and their families have the responsibility for detecting potential test-related problems in the home setting." This section identifies key areas for assessment and provides instruction for what to do when problems are detected.

Abnormal finding: Abnormally high and low values are listed where appropriate.

A blank space is included at the end of the last section for each test, for individualizing the studies to the institution performing the test.

This reference manual is appropriate for use in respiratory therapy programs when students are in the hospital on clinical rotation. The book is small enough to be easily slipped into the pocket of a uniform jacket, where it would be invaluable to the student clinician. The book would be very useful for respiratory therapy students trying to develop critical thinking skills through problem-based learning. This manual would be a great reference for use in courses that present or are organized around clinical cases where large numbers of laboratory tests and laboratory procedures must be understood and interpreted.

During my review of this manual only a few limitations were identified. The section on oximetry did not mention false readings due to nail polish, skin pigmentation, or inadequate perfusion during shock. The accuracy range of and the way to correlate the oximeter heart rate with readings by auscultation or electrocardiogram were not presented. The only desaturation threshold mentioned was a possible critical S_{pO_2} [oxygen saturation measured via pulse oximetry] of ≤ 75%. No guidance was given regarding the range of variance for multiple S_{pO_2} readings during spot checks. Under contraindications, no warning is given to not use pulse-oximetry with patients suffering from carbon monoxide poisoning. Under the arterial blood gas analysis entry, the discussion on sampling does not encourage the user to collect blood from the nondominant hand if possible. The additional complications related to gathering a sample at sites other than the radial arteries are not discussed. The arterial blood gas analysis procedure and patient care section states, "note that an arterial puncture is performed by laboratory technicians, respiratory-inhalation therapists. . . ." The authors and publisher should have known that the term "inhalation therapist" was retired 20 years ago. The chest radiograph abnormal findings are listed without any detailed discussion on what may be observed (eg, "diaphragm" has only one listing: diaphragmatic/hiatal hernia) with no discussion of flat diaphragms observed with hyperinflation. Under pulmonary function tests, "functional residual capacity" is described as "functional residual volume," breaking the cardinal pulmonary physiology rule that two or more lung volumes are always described as a capacity.

Overall this is a very well researched and well written reference manual. Despite the limitations mentioned above, the book should be a very useful addition to the personal libraries of respiratory therapy students who are presented with a battery of laboratory and diagnostic procedures that must be understood and interpreted.

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Rapid Interpretation of Ventilator Waveforms. Jonathan B Waugh PhD RRT CPFT, Vijay M Deshpande MS RRT, Robert J Harwood MSA RRT. Upper Saddle River, New Jersey: Prentice Hall. 1999. Soft-cover, illustrated, 132 pages, $30.60.

Many modern intensive care ventilators monitor airway mechanics and display flow, volume, and pressure waveforms. Using ventilator waveforms might improve ventilator management, provided the clinician can make optimal use of this additional information. The aim of **Rapid Interpretation of Ventilator Waveforms** is to provide a more comprehensive and detailed look at ventilator waveform analysis than regular textbooks. The authors emphasize that the book is intended to improve understanding of how ventilator waveforms are generated and how waveforms reflect abnormal conditions during mechanical ventilation. More than 140 waveform samples—conceptual illustrations and real waveform tracings—are presented in 5 chapters and 5 case studies.

The first two chapters cover the basic principles of ventilator waveform analysis. First the reader is made familiar with the basic flow versus time, pressure versus time, and volume-versus time tracings. Starting out with an example, changes in ventilator settings and their corresponding ventilator waveforms are discussed. Several graphs are used to explain the interdependence of respiratory rate, inspiratory flow, and inspiratory to expiratory time, as well as the influence of airway mechanics on pressure-versus-time waveforms. At the end of the first chapter, tracings of commonly employed modes of ventilation are shown in two formats to compare simplified illustrations with original patient tracings. The second chapter examines the pressure-volume and flow-volume relationships of the respiratory system during mechanical ventilation. Illustrations of the static pressure-volume curve, and real-time recordings of corresponding pressure-volume (P-V) and flow-volume loops are used to explain their specific patterns because of changes in compliance and airway resistance. In this context the authors also explain how the P-V loop reflects dynamic compliance, work of breathing, and the meaning of the inflection points on the compliance curve.

The next two chapters are more oriented to the clinical application of waveform analysis. Using real-time recordings, characteristic waveform patterns of the most common ventilation modes and their combination (eg, synchronized intermittent mandatory ventilation with pressure-support ventilation) are discussed. The following chapter presents common clinical situations, such as acute changes in compliance, alveolar overdistention, and detection of a blocked or kinked endotracheal tube. Several tracings are used to discuss the problems caused by increased airway resistance. The assessment of bronchodilator therapy using before-and-after flow-volume and P-V loops is discussed in detail, as well as the detection of dynamic hyperinflation and intrinsic positive end-expiratory pressure. Also covered in this chapter is the detection of patient-ventilator dyssynchrony due to insufficient inspiratory flow or inappropriate trigger sensitivity.

The final chapter addresses special aspects of waveform analysis during ventilation of neonates and small infants. Although some of the information given had already been presented in previous chapters, special problems such as volume loss due to cuffless endotracheal tubes, detection of right mainstem intubation, and accidental extubation are covered in this chapter. Finally, 5 illustrative cases of patients with abnormal ventilator waveforms are presented in the appendix. Since most of the graphs are taken from the previous chapters, the appendix is a good review for the reader.

Rapid Interpretation of Ventilator Waveforms is the only book I know of that is solely dedicated to ventilator waveform analysis. Its major strength lies in the numerous waveform examples and the color printing that makes it easy to distinguish between various waveforms in the same illustration. However, the approach to keep the "descriptions and commentary. . . to a minimum to enhance clarity and readability" (as stated by the authors in the preface) has its shortcomings. Especially some examples in the second chapter suffer from short, misleading, and even inaccurate information. For instance, the definition of the lower inflection point is too short, and the authors do not make clear that the lower inflection point (reflecting alveolar recruitment) is, ideally, taken from a static P-V curve or at least from a P-V loop with very low constant inspiratory flow. I would have also expected that the authors explain the pitfall that during pressure-targeted ventilation the observed "inflection point" on the P-V loop is an artifact and is due to the basic gas delivery pattern of pressure-control ventilation. At that point I also found the only major mistake in the book, where the authors state that "under conditions of normal resistance and compliance the P-V loop of a pressure-targeted breath is very similar to a volume-targeted breath." By definition, the P-V loop during constant-flow ventilation and pressure-targeted ventilation cannot be similar. During pressure-control ventilation most modern ventilators deliver such high inspiratory flows that the set targeted pressure is almost instantaneously reached and maintained until the end of inspiration. Therefore the pressure in the P-V slope will quickly increase, with an "inflection point" close to the set pressure, forming an almost vertical slope, until the end of inspiration. On the other hand, constant flow ventilation will generate a linear increase in pressure and volume, reaching the maximum pressure at the end of the breath.

Another inaccuracy regarding different modes of ventilation occurs for the definition of dynamic compliance, since the given definition is only true for volume-targeted breaths. As already pointed out, the pressure is almost constant during pressure-control ventilation. In that circumstance, dynamic compliance corresponds to pressure and delivered tidal volume only when inspiratory time is long enough that inspiratory flow during pressure control is almost zero.

Overall, the given information covers most of the clinical situations in which waveform analysis will provide additional information. The authors have reached their goal to write a comprehensive workbook that will enable the reader to use the acquired information at the bedside. The quality of illustrations is excellent, the accompanying text is easy to read, but some aspects need to be more accurately presented. **Rapid Interpretation of Ventilator Waveforms** is a valuable reference source about waveform analysis for the respiratory care student, respiratory therapist, and critical care nurse, as well as for physicians caring for mechanically ventilated patients.

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Pocket Companion for Critical Care Nursing. Sheila Melander DSN RN and Linda Bucher DNSc RN. Philadelphia: WB Saunders. 1999. Soft-cover, illustrated, 542 pages, $24.95.

Dr Melander and Dr Bucher are well known to critical care nurses. Their previous work, the lengthy, complex *Critical Care Nursing*, is regarded as a valuable, evidence-based reference and systematic study guide.

The Pocket Companion for Critical Care Nursing, essentially a précis of their previous work, is intended to be a reference pocket volume for use at the bedside by the hands-on practitioner. There is a substantial nursing market for portable, quick-look information texts, in part as a result of the increasing technological and work-load demands for critical care clinicians, and the declining shelf-life of the experienced intensive care unit nurse. The authors of this book, although intending their primary readership to be nurses, also hope to reach other members of multidisciplinary clinical teams, including respiratory therapists and physicians.

The text is therefore wide-ranging in scope and subject matter. Organized around a body-system basis, each of the 7 sections begins with a brief anatomy and physiology review, followed by assessment, diagnosis, and treatment of illnesses commonly encountered in critical care. Prose clarity renders the book easily readable, there are nicely arranged and accurate tables, and there is little overlap throughout—especially admirable in view of the wide range of subject matter in more than 500 densely packed pages. Notable are the diagnostic testing guides, something most practitioners have not fluently memorized. There are particularly useful and coherent sections dealing with human immunodeficiency virus opportunistic infections, simple and mixed acid-base disorders, and a well arranged and easily understood guide for bedside pulmonary measurements and respiratory pharmacology.

There is marked contrast in the implied prerequisite knowledge level and practitioner focus. For example, there is a full photographic page of testing for pitting edema, algorithms of diagnostic guides for blunt and penetrating abdominal trauma, as well as various concrete patient-teaching suggestions. The directions for basic critical care nursing procedures are accurate and clear, but in the context of quick-look bedside needs, perhaps redundant. For example, a detailed 18-step prescription for correct endotracheal tube suctioning focuses on appropriate matters of equipment utilization and a protocol for organized clinical assessment. This is valuable and technically accurate, but of more use for the beginning student than for the daily practitioner in search of a rapid reference.

The book's weight and size preclude carrying it in a scrub pocket, and the lack of tab dividers and the spread of subject text over several pages hinders immediate access of material. The attractive cover does not lie flat when opened and is not laminated; it is therefore difficult to use by the bedside. Additionally, given the range and volume of material, the print is necessarily quite small: for example, the advanced cardiac life support algorithms are tiny and blurred, hard to read under the most tranquil conditions.

Overall, the content is of the high quality and accuracy that is associated with these authors' work, their attempt to produce a précis of their large text not unsuccessful, and, indeed, of potential interest and value to a range of practitioners. However, although criteria for the ideal rapid pocket reference are necessarily somewhat subjective, most nurses are looking for a focus on immediate quick-look information. Frequently sought is easily readable, brief and highly focused information about vasoactive drip rates and concentrations, hemodynamic numbers, ventricular pacing, advanced cardiac life support protocols, and emergency medications, all this in a small waterproof volume that can be put in a pocket, that will survive a day at the bedside, and preferably that has the ability to allow for a degree of individual inserts and rearrangement.

I recommend this volume for less urgent and inconvenient circumstances than the bedside.

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A Dream of the Heart: The Life of John H Gibbon Jr, Father of the Heart-Lung Machine. Harris B Schumacker Jr MD. Santa Barbara: Fithian Press. 1999. Hardcover, illustrated, 301 pages, $24.95.

John H Gibbon, the subject of the book, was known intimately by the author, himself a cardiac surgeon of renown. As such, the author, Schumacker, seeks to describe "in general terms" what Dr Gibbon contributed to cardiothoracic surgery specifically and medicine generally, as well as to describe him "as a person." In both of these the author achieves his aim.

There are several reasons why this is a valid and important undertaking. John Gibbon was born in 1903, into tremendous intellectual and educational potential and promise. During his career he saw the introduction of antibiotics, advances in anesthesia, development of residency training, and the evolution of thoracic surgery, the last hurdle of which was the ability to operate within the heart and support oxygenation mechanically. As such, Gibbon's life might be considered a metaphor for the medical aspects of "The American Century."

The author paints, as he himself describes in the introduction, not a chronological description of the development of either the heart-lung machine or Gibbon's career development, but rather images to allow a fuller description of the man and the creation. In a sense, he achieves a certain Seurat-like painting—one which viewed from a distance is pleasing, but with closer observation one realizes that the portrait is a carefully constructed conglomeration of points. These points are both tantalizing and revealing. We are told that the subject could not relate to his children verbally, but that he had very warm feelings toward them, expressed in nonverbal pursuits. He was considered a kind, gentle, and intellectual man, but also there are hints of pride, rage, and resolve, almost to the point of complete self-absorption.

Gibbon, who wanted to be a poet and artist, married Malay, trained as an artist but who wanted to be a medical researcher. In fact they meet in Dr Churchill's lab. Perhaps it was Gibbon's wistfullness that allowed him to wonder, as he watched a patient labor under the hypoxic stress of a massive pulmonary embolism, about the feasibility of developing extracorporeal circulation. Certainly, he seems to be more intrigued by the physiologic problem than the device itself. Having demonstrated its effectiveness, he turned his attention to an entirely different arena.

The author does achieve his aims of describing in general terms the history of the development of extracorporeal circulation. He specifically avoids technical details that are well-described elsewhere, and instead focuses on how Dr Gibbon's overall style of research allowed him to concentrate on the development of the machine based on both his own and others' research. It might have been helpful to review briefly, but in more detail, some key research work done by Dr Gibbon, as illustration of where he was at particular times. Dr Schumacker describes with great enjoyment the contributions of both the subject of his narrative and of others in the field. When one realizes the diverse personalities (such as Charles Lindbergh and Alexis Carrel) who were involved in studying the problem, it is clear that this is a story that should be savored!

Some may find the book difficult to read. The author "leaps about," with earlier chapters describing Gibbon's lineage, particularly as far as the Confederate side is concerned, with later descriptions of his great uncle, John Gibbon, leader of the union "Black Hat Brigade" (also recognized by Stonewall Jackson's men as "those damn blackhat fellers" after a stiff engagement).[1] There is only one egregious error, in my mind. This occurs when, describing General Gibbon's role at Gettysburg, the author attributes General Sickle's ludicrous maneuver, pushing III Corp into the peach orchard, to "Hancock the Magnificent." The author also dwells excessively on the numerous awards and degrees that were given to Dr Gibbon. It would have been better to list them in an appendix and select a few important ones to describe their importance in better detail.

In the end, there are several reasons to read this book. Anyone with an interest in mechanical circulatory support (including thoracic surgeons, perfusionists, cardiologists, and pulmonary intensivists) will enjoy the developmental stages described. Young surgical investigators and older established educators will see parallels in the struggle to perform good research, based on "the question" rather than "the anticipated result." Educators will recognize the struggle that Gibbon foresaw in encouraging academic surgeons to dedicate time to research and teaching. All physicians will be interested in the observations Gibbon made predicting the pros and cons regarding health maintenance organizations.

If Gibbon had never worked on the heart lung machine, his achievements in education, teaching, and research would still be impressive. One wonders if current administrators and chairmen appreciate the value of good science. Young surgeons considering embarking on an academic career should consider the trials and tribulations this may cause for family life. Finally, the lay public would enjoy reading about a man who exemplified all that is best about medical care and research. Like Jonas Salk, John Gibbon refused to profit from his discoveries and did not patent the bypass machine. In the end, Harris Schumacker leaves us wondering, wistfully, if John Gibbon was "one of the few good men," the like of which are rarer and rarer.

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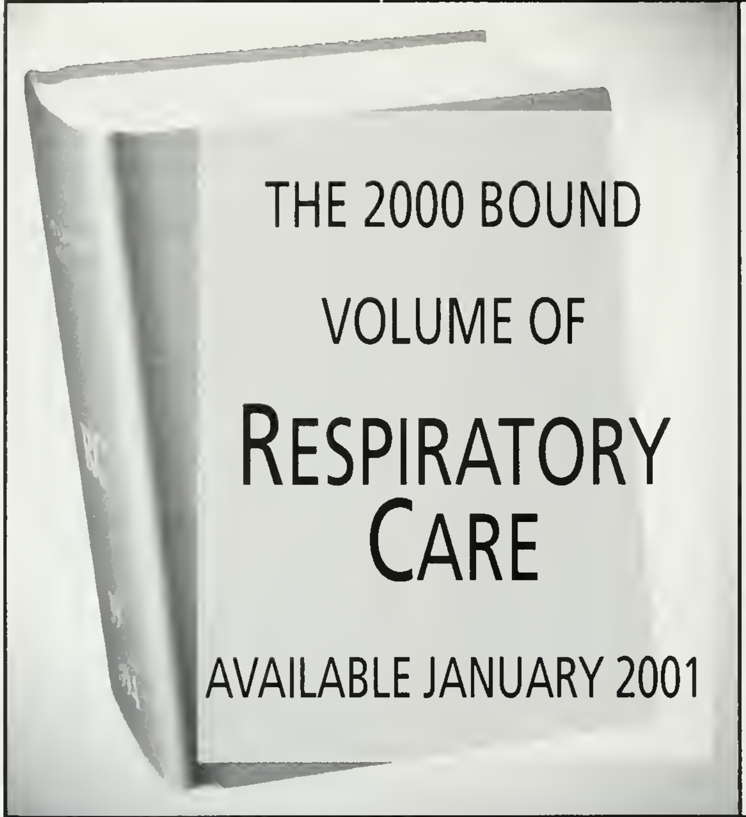
REFERENCE

1. Sears SW. Landscape turned red: the battle of Antietam. Boston: Houghton Mifflin; 1983:156.



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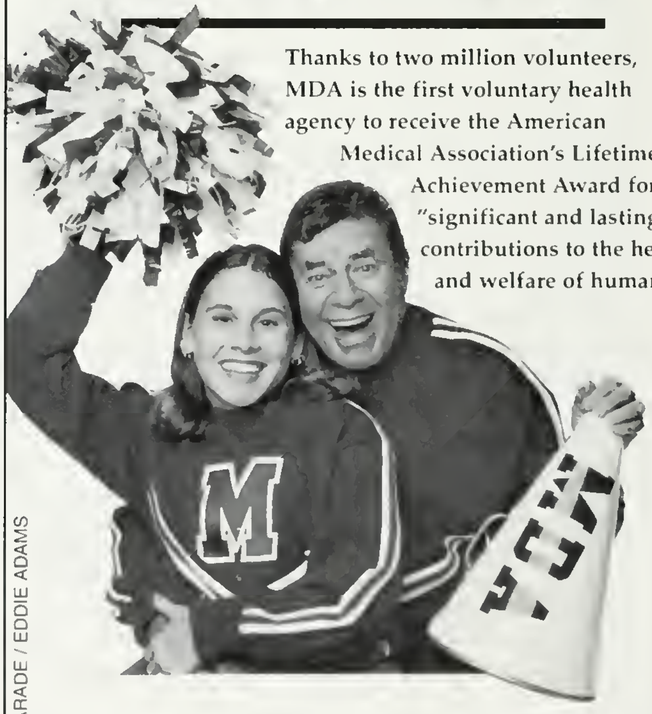

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I hereby apply for membership in the American Association for Respiratory Care and have enclosed my dues. If approved for membership in the AARC, I will abide by its bylaws and professional code of ethics. I authorize investigation of all statements contained herein and understand that misrepresentations or omissions of facts called for is cause for rejection or expulsion.

A yearly subscription to RESPIRATORY CARE journal and *AARC Times* magazine includes an allocation of $11.50 from my dues for each of these publications.

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call for abstracts

RESPIRATORY CARE • OPEN FORUM 2001

The American Association for Respiratory Care and its science journal, RESPIRATORY CARE, invite submission of brief abstracts related to any aspect of cardiorespiratory care. The abstracts will be reviewed, and selected authors will be invited to present posters at the OPEN FORUM during the AARC International Respiratory Congress in San Antonio, Texas, December 1-4, 2001. Accepted abstracts will be published in the October 2001 issue of RESPIRATORY CARE. Membership in the AARC is not required for participation. All accepted abstracts are automatically considered for ARCF research grants.

SPECIFICATIONS—READ CAREFULLY!

An abstract may report **(1) an original study, (2) the evaluation of a method, device or protocol, or (3) a case or case series.** Topics may be aspects of adult acute care, continuing care/rehabilitation, perinatology/pediatrics, cardiopulmonary technology, or health care delivery. The abstract may have been presented previously at a local or regional—but not national—meeting and should not have been published previously in a national journal. The abstract will be the only evidence by which the reviewers can decide whether the author should be invited to present a poster at the OPEN FORUM. Therefore, *the abstract must provide all important data, findings, and conclusions.* Give specific information. Do not write such general statements as "Results will be presented" or "Significance will be discussed."

ESSENTIAL CONTENT ELEMENTS

Original study. Abstract *must* include (1) Background: statement of research problem, question, or hypothesis; (2) Method: description of research design and conduct in sufficient detail to permit judgment of validity; (3) Results: statement of research findings with quantitative data and statistical analysis; (4) Conclusions: interpretation of the meaning of the results.

Method, device, or protocal valuation. Abstract *must* include (1) Background: identification of the method, device, or protocol and its intended function; (2) Method: description of the evaluation in sufficient detail to permit judgment of its objectivity and validity; (3) Results: findings of the evaluation; (4) Experience: summary of the author's practical experience or a lack of experience; (5) Conclusions: interpretation of the evaluation and experience. Cost comparisons should be included where possible and appropriate.

Case report. Abstract *must* report a case that is uncommon or of exceptional educational value and must include (1) Introduction: relevant basic information important to understanding the case. (2) Case Summary: patient data and response, details of interventions. (3) Discussion: content should reflect results of literature review. The author(s) should have been actively involved in the case and a case-managing physician must be a co-author or must approve the report.

FORMAT AND TYPING INSTRUCTIONS

Accepted abstracts will be photographed and reduced by 40%; therefore, the size of the original text should be *at least* 10 points. A font like Helvetica or Times makes the clearest reproduction. The first line of the abstract should be the title in all capital letters. Title should explain content. Follow title with names of all authors (including credentials), institution(s), and location; underline presenter's name. Type or electronically print the abstract *single spaced in one paragraph on a clean sheet of paper, using margins set so that the abstract will fit into a box no bigger than 18.8 cm (7.4") by 13.9 cm (5.5"), as shown on the reverse of this page.* Insert only one letter space between sentences. Text submission on diskette is allowed but must be accompanied by a hard copy. *Data may be submitted in table form, and simple figures may be included provided they fit within the space allotted. No figure, illustration, or table is to be attached to the abstract form.* Provide all author information requested. Standard abbreviations may be employed without explanation; new or infrequently used abbreviations should be spelled out on first use. Any recurring phrase or expression may be abbreviated, if it is first explained. Check the abstract for (1) errors in spelling, grammar, facts, and figures; (2) clarity of language; and (3) conformance to these specifications. An abstract not prepared as requested may not be reviewed. Questions about abstract preparation may be telephoned to Linda Barcus at (972) 406-4667.

Early Deadline Allowing Revision. Authors may choose to submit abstracts early. Abstracts postmarked by May 31, 2001 will be reviewed and the authors notified *by letter only* to be mailed by June 15, 2001. Rejected abstracts will be accompanied by a written critique that should, in many cases, enable authors to revise their abstracts and resubmit them by the Final Deadline (July 17, 2001).

Final Deadline. The mandatory Final Deadline is July 17, 2001 (postmark). Authors will be notified of acceptance or rejection *by letter only.* These letters will be mailed by September 1, 2001.

Mailing Instructions. Mail (Do not fax!) 2 clear copies of the completed abstract form, diskette (if possible), and a stamped, self-addressed postcard (for notice of receipt) to:

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Categories of Articles

Research Article: A report of an original investigation (a study). Must include Title Page, Abstract, Key Words, Background, Methods, Results, Discussion, Conclusions, and References. May also include Tables, Figures (if so, must include Figure Legends), Acknowledgments, and Appendices.

Review Article: A comprehensive, critical review of the literature and state-of-the-art summary of a topic that has been the subject of at least 40 published research articles. Must include: Title Page, Outline, Key Words, Introduction, Review of the Literature, Summary, and References. May also include: Tables, Figures (if so, must include Figure Legends), and Acknowledgments.

Overview: A critical review of a pertinent topic that has fewer than 40 published research articles. Same structure as Review Article.

Update: A report of subsequent developments in a topic that has been critically reviewed in RESPIRATORY CARE or elsewhere. Same structure as a Review Article.

Special Article: A pertinent paper not fitting one of the other categories. Consult with the Editor before writing or submitting such a paper.

Editorial: A paper addressing an issue in the practice or administration of respiratory care. It may present an opposing opinion, clarify a position, or bring a problem into focus.

Letter: A brief, signed communication responding to an item published in RESPIRATORY CARE or about other pertinent topics. Tables, Figures, and References may be included. The letter should be marked "For Publication."

Case Report: Report of an uncommon clinical case or a new or improved method of management or treatment. A case-managing physician must either be an author or furnish a letter approving the manuscript. Must include: Title Page, Abstract, Introduction, Case Summary, Discussion, and References. May also include: Tables, Figures (if so, must include Figure Legends), and Acknowledgments.

Point-of-View Paper: A paper expressing personal but substantiated opinions on a pertinent topic. Must include: Title Page, Text, and References. May also include Tables and Figures (if so, must include Figure Legends).

Drug Capsule: A miniature review paper about a drug or class of drugs that includes discussions of pharmacology, pharmacokinetics, or pharmacotherapy.

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> Rau JL, Harwood RJ. Comparison of nebulizer delivery methods through a neonatal endotracheal tube: a bench study. Respir Care 1992;37(11):1233-1240.

Article in a publication that numbers each issue beginning with Page 1:

> Bunch D. Establishing a national database for home care. AARC Times 1991;15(Mar):61,62,64.

Corporate author journal article:

> American Association for Respiratory Care. Criteria for establishing units for chronic ventilator-dependent patients in hospitals. Respir Care 1988;33(11):1044-1046.

Article in journal supplement: (Journals differ in numbering and identifying supplements. Supply information sufficient to allow retrieval.)

> Reynolds HY. Idiopathic interstitial pulmonary fibrosis. Chest 1986; 89(3 Suppl):139S-143S.

Abstract in journal: (Abstracts citations are to be avoided, and those more than 3 years old should not be cited.)

> Stevens DP. Scavenging ribavirin from an oxygen hood to reduce environmental exposure (abstract). Respir Care 1990;35(11): 1087-1088.

Editorial in a journal:

> Enright P. Can we relax during spirometry? (editorial). Am Rev Respir Dis 1993;148(2):274.

Editorial with no author given:

> Negative-pressure ventilation for chronic obstructive pulmonary disease (editorial). Lancet 1992;340(8833):1440-1441.

Letter in journal:

> Aelony Y. Ethnic norms for pulmonary function tests (letter). Chest 1991;99(4):1051.

Corporate author book:

> American Medical Association Department of Drugs. AMA drug evaluations, 3rd ed. Littleton CO: Publishing Sciences Group; 1977.

Book: (For any book, specific pages should be cited whenever reference is made to specific statements or other content.)

> DeRemee RA. Clinical profiles of diffuse interstitial pulmonary disease. New York: Futura; 1990:76-85.

Chapter in book with editor(s):

> Pierce AK. Acute respiratory failure. In: Guenter CA, Welch MH, editors. Pulmonary medicine. Philadelphia: JB Lippincott; 1977:26-42.

Paper accepted but not yet published:

> Hess D. New therapies for asthma. Respir Care (year, in press).

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New Products & Services

Digital Handheld Pulse Oximeter. Nonin Medical Inc has released the newest addition to its digital pulse oximeter product line—the PalmSAT™ 2500. Nonin describes the new device as lightweight and versatile and says that it is designed to accurately assess blood oxygen saturation and pulse rate. According to the company, the PalmSAT 2500 is appropriate for use in multiple care settings and offers 72 hours of memory standard, ergonomic design, simple operation, and convenient battery access. For more information from Nonin Medical Inc, circle number 191 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

Ventilatory Support System. Respironics Inc has released its new BiPAP® Synchrony™ Ventilatory Support System. The company says this newest member of its BiPAP line offers providers the option of using either a limited-feature ventilator platform (BiPAP Synchrony S) or an advanced platform (BiPAP Synchrony S/T) with additional modes of operation. Respironics says the device is lightweight at less than 6 pounds and that it offers patient comfort features such as Digital Auto-Trak Sensitivity™, which automatically adjusts to a patient's breathing pattern and mask leaks, and a ventilator ramp function that allows pressure to gradually be introduced to the patient. For more information from Respironics Inc, circle number 192 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

Spirometer/Pulse Oximeter Combination. QRS Diagnostic received 510(k) clearance from the Food and Drug Administration to market what the company calls the world's first combination spirometer/pulse oximeter. QRS says the SpirOxCard®, available since late 2000, provides two sophisticated diagnostic tests with only one device, noting also that it is small enough to fit in a coat pocket and that it is compatible with most Windows® CE handheld computers. The company also says the new device, along with the company's Office Medic software, will cost significantly less than competitive, stand-alone systems. For more information from QRS Diagnostic circle number 193 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

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March 14–16	22nd Annual Tahoe Conference, **Lake Tahoe, CA**	CSRC Greater Bay Area Chapter, (925) 866-6643; www.cssrc.org
March 27	Professor's Rounds 2001 Live Videoconference, Program 2	AARC, (972) 243-2272
April 4–5	USRC 2001 Conference; **Ogden, UT**	Utah Society for Respiratory Care, peberele@weber.edu
April 19–20	PSRC 4th Annual Eastern Regional Conference and Exhibition, **King of Prussia, PA**	Pennsylvania Society for Respiratory Care, Ann Cusano, (215) 646-7300, ext. 428, cusano.a@gmc.edu
July 21–23	Management and Education Sections, Summer Forum; **Naples, FL**	AARC, (972) 243-2272, clay@aarc.org
Dec 1-4	47th International Respiratory Congress; **San Antonio, TX**	AARC, (972) 243-2272, clay@aarc.org

Date	Other Meetings	Contact
March 16–17	UT Health Science Center and Wilford Hall USAF Medical Center's 6th Annual Respiratory Care Symposium; **San Antonio, TX**	UTHSCSA, Dept. of Respiratory Care, (210) 567-8850
May 1	Spirometry Update Refresher Course, **Cincinnati, OH**	Dr. Roy T. McKay, (513) 558-1234 or www.drmckay.com
May 8-11	All Children's Hospital: Neonatal/Pediatric Transport Conference, **Clearwater Beach, FL**	Connie Spadaccino, (800) 456-4543, ext. 4240
May 12–14	Spring Sleep Seminar 2001, **Branson, MO**	Bill Rivers or Melinda Trimble, (501) 713-1272

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2001 RESPIRATORY CARE OPEN FORUM

Early Deadline: May 31, 2001
Final Deadline: July 17, 2001

Notices

Notices of competitions, scholarships, fellowships, examination dates, new educational programs, and the like will be listed here free of charge. Items for the Notices section must reach the Journal 60 days before the desired month of publication (January 1 for the March issue, February 1 for the April issue, etc). Include all pertinent information and mail notices to RESPIRATORY CARE Notices Dept, 11030 Ables Lane, Dallas TX 75229-4593

Scheduled Professor's Rounds 2001

Program #1 Taking the Mystery Out of Ventilator Weaning for Children—Peter Betit BS RRT FAARC; Host Richard D Branson BA RRT FAARC—Video March 13 Audio April 10

Program #2 Pulmonary Rehabilitation: Standard Care for Chronic Lung Disease Patients—Trina Limberg BS RRT; Host Thomas J Kallstrom RRT FAARC—Video March 27 Audio April 17

Program #3 Noninvasive Ventilation: The Latest Word—Dean R Hess PhD RRT FAARC; Host Richard D Branson BA RRT FAARC—Video April 24 Audio May 29

Program #4 Patient Education for the Asthmatic—Tracey Mitchell RRT; Host Thomas J Kallstrom RRT FAARC—Video May 22 Audio June 19

Program #5 ARDS: The Disease and Its Management—Leonard D Hudson MD; Host David J Pierson MD FAARC—Video June 26 Audio July 17

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Program #7 Invasive Ventilation: The Latest Word—Richard H Kallet MS RRT; Host Richard D Branson BA RRT FAARC—Video September 25 Audio October 16

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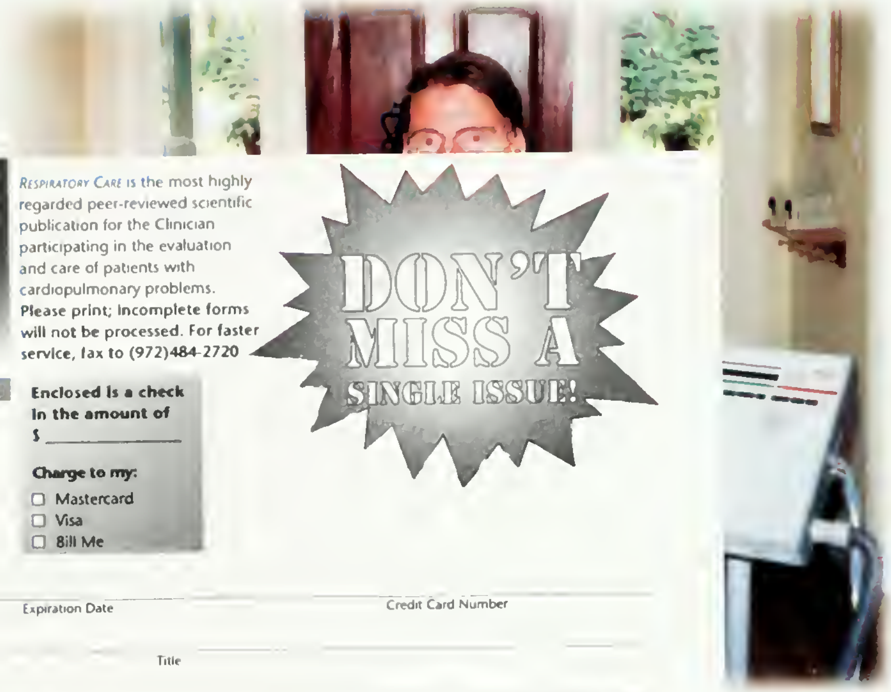

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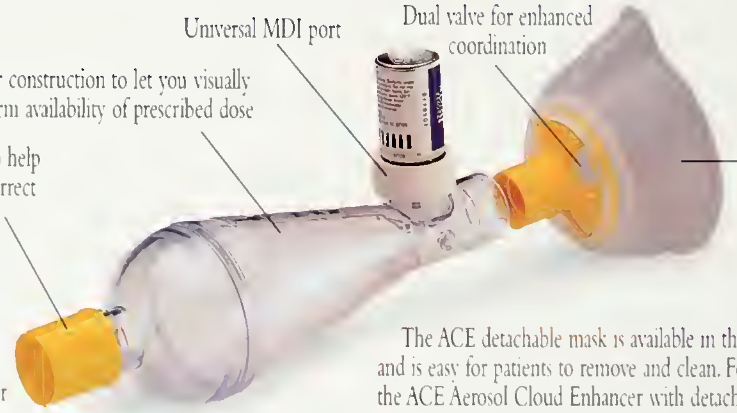
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